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Requester's Full Name: William J. Miller Examiner #: 1111 Date: 5/16/93
 Art Unit: 254 Phone Number 30 1111 Serial Number: 1111
 Mail Box and Bldg/Room Location: 1111 Results Format Preferred (circle): PAPER DISK E-MAIL

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Please provide a detailed statement of the search topic, and describe as specifically as possible the subject matter to be searched. Include the elected species or structures, keywords, synonyms, acronyms, and registry numbers, and combine with the concept or utility of the invention. Define any terms that may have a special meaning. Give examples or relevant citations, authors, etc. if known. Please attach a copy of the cover sheet, pertinent claims, and abstract

Title of Invention: PROSTATE GLAND

Inventors (please provide full names): W. J. Miller

Earliest Priority Filing Date: 5/16/93

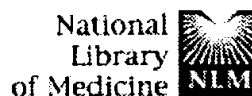
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Clerical Prep Time: <u>1111</u>	Patent Family <u>1111</u>	WWW/Internet <u>1111</u>
Online Time: <u>1111</u>	Other <u>1111</u>	Other (specify) <u>1111</u>



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Protein

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#9 Search mercaptopropanoic and desmopressin	10:14:04	<u>2</u>
#8 Search mercaptopropanoic and water	10:13:45	<u>1</u>
#7 Search mercaptopropanoic	10:13:28	<u>27</u>
#6 Search mercapto and propanyl	10:13:14	<u>0</u>
#5 Search mercaptopropanyl	10:13:05	<u>0</u>
#4 Related Articles for PubMed (Select 926117)	10:02:55	
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May 2 2003 16:34:23

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STRUCTURE FILE UPDATES: 15 MAY 2003 HIGHEST RN 516445-69-5
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TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2003

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(FILE 'HOME' ENTERED AT 11:43:04 ON 16 MAY 2003)

FILE 'REGISTRY' ENTERED AT 11:43:41 ON 16 MAY 2003
 E IGF-1

L1 64 S E4-E6
 L2 1073 S INSULIN(L)GROWTH(L)FACTOR(L) (1 OR ONE OR I)
 L3 1128 S L1 OR L2

FILE 'HCAPLUS' ENTERED AT 11:44:37 ON 16 MAY 2003

L4 20729 S L3 OR ((INSULIN(W)LIKE OR INSULIN) (W)GROWTH(W)FACTOR? OR IGF
 L5 1485 S L4(L)CYCL?
 L6 31 S L4(2N)CYCLIC?
 SELECT HIT RN L6 1-31

FILE 'REGISTRY' ENTERED AT 11:47:47 ON 16 MAY 2003

L7 2 S E1-E3

FILE 'REGISTRY' ENTERED AT 11:50:19 ON 16 MAY 2003

=> d sqide 17 1-2

L7 ANSWER 1 OF 2 REGISTRY COPYRIGHT 2003 ACS

RN **122635-51-2** REGISTRY

CN Insulin-like growth factor I (human reduced), cyclic
 (6.fwdarw.47), (18.fwdarw.61), (48.fwdarw.52)-tris(disulfide) (9CI) (CA
 INDEX NAME)

OTHER NAMES:

CN Cyclic (6.fwdarw.47), (18.fwdarw.61), (48.fwdarw.52)-tris(disulfide) human
 IGF-I

CN Human insulin-like growth factor-I, isomer I

CN Insulin-like growth factor I (human improperly folded isoform)

CN Insulin-like growth factor I (human isoform)

FS PROTEIN SEQUENCE

SQL 70

NTE

type	location	description
bridge	Cys-6 - Cys-47	disulfide bridge
bridge	Cys-18 - Cys-61	disulfide bridge
bridge	Cys-48 - Cys-52	disulfide bridge

SEQ 1 GPETLCGAEL VDALQFVCGD RGFYFNKPTG YGSSSRAPQ TGIVDECCFR
51 SCDLRRLEMY CAPLKPAKSA

RELATED SEQUENCES AVAILABLE WITH SEQLINK

DR 157320-42-8
MF C331 H512 N94 O101 S7
CI MAN
SR CA
LC STN Files: CA, CAPLUS
11 REFERENCES IN FILE CA (1957 TO DATE)
11 REFERENCES IN FILE CAPLUS (1957 TO DATE)

L7 ANSWER 2 OF 2 REGISTRY COPYRIGHT 2003 ACS

RN **67763-96-6** REGISTRY

CN Insulin-like growth factor 1 (9CI) (CA INDEX NAME)

OTHER NAMES:

CN IGF-1
CN IGF-I
CN Insulin-like growth factor 1
CN insulin-like growth factor I
CN Somatomedin 1
CN Somatomedin C
CN Sulfation factor C
DR **61461-67-4**
MF Unspecified
CI PMS, COM, MAN
PCT Manual registration
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BIOBUSINESS,
BIOSIS, BIOTECHNO, CA, CANCERLIT, CAPLUS, CBNB, CEN, CHEMCATS, CIN,
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*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***

15377 REFERENCES IN FILE CA (1957 TO DATE)
271 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
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L6 ANSWER 1 OF 31 HCAPLUS COPYRIGHT 2003 ACS
ACCESSION NUMBER: 2001:101108 HCAPLUS
DOCUMENT NUMBER: 134:141764
TITLE: Cyclic amine derivatives for the treatment of neurological diseases
INVENTOR(S): Mullican, Michael; Lauffer, David
PATENT ASSIGNEE(S): Vertex Pharmaceuticals Incorporated, USA
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001009097	A1	20010208	WO 2000-US18578	20000706
<p>W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM</p> <p>RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG</p>				
EP 1202970	A1	20020508	EP 2000-947092	20000706
<p>R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL</p>				
JP 2003506356	T2	20030218	JP 2001-514301	20000706
US 2002123493	A1	20020905	US 2002-40033	20020103
PRIORITY APPLN. INFO.:			US 1999-146588P	P 19990730
			WO 2000-US18578	W 20000706
OTHER SOURCE(S): MARPAT 134:141764				
<p>AB The present invention relates to cyclic amine derivs. of general formula (I) for treating or preventing neuronal damage assocd. with neurol. diseases. The invention also provides compns. comprising the compds. of the present invention and methods of utilizing those compns. for treating or preventing neuronal damage. The invention also includes use of the compds. in combination with neurotrophic factors.</p>				
<p>IT 67763-96-6, IGF-1</p> <p>RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)</p> <p>(cyclic amine derivs. for treatment of neurol. diseases and their use in combination with neurotrophic factors)</p>				

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001008685	A1	20010208	WO 2000-US20491	20000727
W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
EP 1207882	A1	20020529	EP 2000-952238	20000727
R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL			
JP 2003505508	T2	20030212	JP 2001-513415	20000727
PRIORITY APPLN. INFO.:			US 1999-146582P P	19990730
			WO 2000-US20491 W	20000727

OTHER SOURCE(S): MARPAT 134:141761

AB The present invention relates to acyclic and cyclic amine derivs. for treating or preventing neuronal damage assocd. with neurol. diseases. The invention also provides compns. comprising the compds. of the present invention and methods of utilizing those compns. for treating or preventing neuronal damage. The invention includes the use of neurotrophic factors in combination with the acyclic and cyclic amines.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

Page 4

LANGUAGE: English

AB Insulin-like growth factor I (IGF-I) is a potent mitogen for both normal and transformed mammary epithelial cells (MEC), and IGF-binding protein-3 (IGFBP-3) potentiates IGF-I action in these cells. The synthesis of IGFBP-3 is stimulated by both IGF-I and agents that increase intracellular cAMP (e.g. forskolin) in the bovine MEC line MAC-T. In addn., the combination of IGF-I and cAMP increases IGFBP-3 mRNA to a greater extent than does either treatment alone. The mol. mechanisms responsible for this regulation are not known and therefore represent the focus of this study. The half-life of IGFBP-3 mRNA in untreated MAC-T cells was detd. to be 11 h. Exposure to IGF-I or forskolin increased the half-life to 27 and 101 h, resp. Nuclear run-on assays indicated that IGFBP-3 transcription rates were increased 3.5-fold in cells treated with a combination of IGF-I and forskolin. To further study this regulation, 1.1 kb of the 5'-flanking region of the IGFBP-3 promoter were fused to a promoter-less reporter plasmid encoding luciferase. Transient transfection assays indicated that both IGF-I and forskolin alone produced small, but significant, increases in IGFBP-3 promoter activity of 1.57- and 1.59-fold, resp. However, the combination of IGF-I and forskolin increased IGFBP-3 promoter activity 2.25-fold above control values, suggesting that these factors activate discrete signaling pathways that act in concert to stimulate IGFBP-3 gene transcription. Deletion anal. indicated that promoter fragments contg. as little as 267 bp upstream of the TATA box retained responsiveness of IGF-I and forskolin. This region contains a 200-bp sequence that is approx. 80% homologous between the murine and bovine promoters. It contains several conserved AP-2 and Sp1 consensus binding sequences that may be important for the effects of IGF-I and forskolin on IGFBP-3 promoter activity. In summary, these data indicate that IGF-I and cAMP, working through sep. signaling pathways, activate both transcriptional and post-transcriptional mechanisms to stimulate IGFBP-3 synthesis in MEC.

REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 4 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:757391 HCAPLUS

DOCUMENT NUMBER: 133:345122

TITLE: Regulation of insulin-like growth factor-binding protein 1 by hypoxia and 3',5'-cyclic adenosine monophosphate is additive in HepG2 cells

AUTHOR(S): Sugawara, Junichi; Tazuke, Salli I.; Lii, F-Suen; Powell, David R.; Kaper, Fiona; Giaccia, Amato J.; Giudice, Linda C.

CORPORATE SOURCE: Departments of Gynecology and Obstetrics, Stanford University Medical School, Stanford, CA, 94305-5317, USA

SOURCE: Journal of Clinical Endocrinology and Metabolism (2000), 85(10), 3821-3827
CODEN: JCEMAZ; ISSN: 0021-972X

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Insulin-like growth factor-binding protein 1 (IGFBP-1) is important in regulating minute-to-minute IGF bioavailability in the circulation and is primarily an inhibitor of IGF action systemically and in most cellular systems. Understanding regulation of IGFBP-1 is, thus, important in understanding regulation of IGF actions. The IGFBP-1 promoter contains a cAMP response element, and cAMP stimulates IGFBP-1 gene expression at the transcriptional level. Recently, the authors have found three consensus sequences for the hypoxia response element in intron 1 of the IGFBP-1 gene. Herein, the authors have investigated the effects of hypoxia and a cAMP analog, 8-bromoadenosine-3',5'-cyclic monophosphate (8-Br-cAMP), on IGFBP-1 expression in HepG2 cells, a model system for IGFBP-1 gene

regulation. HepG2 cells were exposed to normoxia (20% pO₂) or hypoxia (2% pO₂) for 24 h in the absence or presence of 8-Br-cAMP (0.1, 0.5, and 1 mM). Western ligand blotting revealed IGFBP-1 as the predominant IGFBP in HepG2-conditioned media, which increased in a dose-dependent manner after incubation with 8-Br-cAMP in normoxia and hypoxia (3-fold and 7-fold at 1 mM, resp.). Under hypoxic, compared with normoxic, conditions, IGFBP-1 protein and mRNA levels increased .apprx. 10-fold and 20-fold, resp. In normoxia, 8-Br-cAMP stimulated IGFBP-1 protein and mRNA levels in a dose-dependent manner (7-fold and 10-fold at 1 mM). Hypoxia and 8-Br-cAMP showed additive stimulatory effects on IGFBP-1 protein and mRNA levels (35-fold and 50-fold at 1 mM) that were time and dose dependent. Primary transcripts of IGFBP-1 mRNA were increased concordantly with IGFBP-1 mRNA. The half-life of the IGFBP-1 mRNA was markedly increased (.apprx.6-fold) by hypoxia, and cAMP minimally enhanced this effect. These results demonstrate that hypoxia and compds. that increase intracellular cAMP additively regulate IGFBP-1 gene expression by transcriptional and posttranscriptional mechanisms. Regulation of IGFBP-1 mRNA and protein by cAMP and hypoxia may be important for understanding the physiol. and pathophysiol. roles of IGFBP-1.

REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 5 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:646433 HCAPLUS

DOCUMENT NUMBER: 133:276581

TITLE: Presumptive mediators of growth hormone action on insulin-like growth factor I release by porcine ovarian granulosa cells

AUTHOR(S): Makarevich, A. V.; Sirotkin, A. V.

CORPORATE SOURCE: Research Institute of Animal Production, Nitra, SK-94 992, Slovakia

SOURCE: Biological Signals and Receptors (2000), 9(5), 248-254
CODEN: BSREF3; ISSN: 1422-4933

PUBLISHER: S. Karger AG

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The role of cAMP/protein kinase A (PKA)- and tyrosine kinase (TK)-dependent intracellular mechanisms in mediating the action of porcine growth hormone (GH) on insulin-like growth factor I (IGF-I) secretion by porcine ovarian granulosa cells was studied. It was obsd. that GH-induced stimulation of IGF-I secretion was accompanied by an increase in cAMP prodn. The stimulation of PKA by the addn. of either a cAMP agonist or a phosphodiesterase inhibitor to the medium increased IGF-I release by the cells, indicating a direct stimulation of **IGF-I** release by **cyclic** nucleotides. Moreover, the stimulatory effect of GH on IGF-I was completely suppressed by the addn. of the PKA blocker Rp-cAMPS. Neither TK blocker altered the basal IGF-I level, but both strongly suppressed the GH-induced increase in IGF-I accumulation. Taken together, these findings suggest that cAMP/PKA- and/or TK-dependent pathways may be involved in the mediation of GH action on IGF-I release by porcine granulosa cells.

REFERENCE COUNT: 28 THERE ARE 28 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 6 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:584368 HCAPLUS

DOCUMENT NUMBER: 133:218129

TITLE: The role of IGF-I, cAMP/protein kinase A and MAP-kinase in the control of steroid secretion, cyclic nucleotide production, granulosa cell proliferation and preimplantation embryo development in rabbits

AUTHOR(S): Makarevich, A.; Sirotkin, A.; Chrenek, P.; Bulla, J.; Hetenyi, L.

CORPORATE SOURCE: Research Institute of Animal Production, Nitra, 94992, Slovakia
SOURCE: Journal of Steroid Biochemistry and Molecular Biology (2000), 73(3-4), 123-133
CODEN: JSBBEZ; ISSN: 0960-0760
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The aim of this study was to investigate the actions of insulin-like growth factor I (IGF-I) on the secretory and proliferative functions of rabbit ovarian cells and on early embryogenesis. It was found that addn. of IGF-I at a lower concn. (1 ng/mL) stimulated progesterone secretion by cultured rabbit granulosa cells, while higher concns. of IGF-I (10, 100 ng/mL) were inhibitory. IGF-I had no effect on estradiol secretion. CAMP secretion was slightly increased after addn. of IGF-I at 10 ng/mL, but not by higher concns. Cyclic GMP secretion was stimulated by IGF-I at 100 ng/mL only. A blocker of protein kinase A, Rp-cAMPS, did not alter progesterone and estradiol secretion but did prevent the action of IGF-I on progesterone secretion. An immunocytochem. study demonstrated that IGF-I significantly increased the proportion of proliferating cell nuclear antigen-pos. (PCNA-pos.) cells. Rp-cAMP did not change cell proliferation but partially prevented the proliferation-stimulating effect of IGF-I. IGF-I (100 ng/mL) significantly increased the proportion of divided zygotes and the no. of embryos reaching the morula/blastocyst stage. Blockers of PKA, Rp-cAMPS and KT5720, reversed the effects of IGF-I on zygote cleavage and embryo development. Addn. of IGF-I (100 ng/mL) significantly increased MAPK within the cells (proportion showing immunoreactivity to ERK-1 and ERK-3 antibodies and intensity of a 42-kDa band related to ERK-2). Rp-cAMPS suppressed the basal ERK-2 immunoreactivity but not that of ERK-1 or ERK-3. It completely inhibited the IGF-I-induced activation of ERK-3 but not that of ERK-1 or ERK-2. This in vitro study demonstrates that IGF-I is a potent stimulator of ovarian secretion, proliferation and embryogenesis in rabbit. Its effects are mediated by cAMP/PKA- and, probably by, MAPK-dependent intracellular mechanisms.

REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 7 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:291572 HCAPLUS

DOCUMENT NUMBER: 133:57537

TITLE: Cyclic nucleotide phosphodiesterase 3B is a downstream target of protein kinase B and may be involved in regulation of effects of protein kinase B on thymidine incorporation in FDCP2 cells

AUTHOR(S): Ahmad, Faiyaz; Cong, Li-Na; Holst, Lena Stenson; Wang, Ling-Mei; Rahn Landstrom, Tova; Pierce, Jaclyn H.; Quon, Michael J.; Degerman, Eva; Manganiello, Vincent C.

CORPORATE SOURCE: Pulmonary/Critical Care Medicine Branch, National Institutes of Health, Bethesda, MD, 20892, USA

SOURCE: Journal of Immunology (2000), 164(9), 4678-4688

CODEN: JOIMA3; ISSN: 0022-1767

PUBLISHER: American Association of Immunologists

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Wild-type (F/B), constitutively active (F/B*), and 3 kinase-inactive (F/Ba-, F/Bb-, F/Bc-) forms of Akt/protein kinase B (PKB) were permanently overexpressed in FDCP2 cells. In the absence of insulin-like growth factor-1 (IGF-1), activities of PKB, cyclic nucleotide phosphodiesterase 3B (PDE3B), and PDE4 were similar in nontransfected FDCP2 cells, mock-transfected (F/V) cells, and F/B and F/B- cells. In F/V cells, IGF-1 increased PKB, PDE3B, and PDE4 activities approx.2-fold. In F/B cells,

IGF-1, in a wortmannin-sensitive manner, increased PKB activity .apprx.10-fold and PDE3B phosphorylation and activity (.apprx.4-fold), but increased PDE4 to the same extent as in F/V cells. In F/B* cells, in the absence of IGF-1, PKB activity was markedly increased (.apprx.10-fold) and PDE3B was phosphorylated and activated (3-4-fold); wortmannin inhibited these effects. In F/B* cells, IGF-1 had little further effect on PKB and activation/phosphorylation of PDE3B. In F/B- cells, IGF-1 activated PDE4, not PDE3B, suggesting that kinase-inactive PKB behaved as a dominant neg. with respect to PDE3B activation. Thymidine incorporation was greater in F/B* cells than in F/V cells and was inhibited to a greater extent by PDE3 inhibitors than by rolipram, a PDE4 inhibitor. In F/B cells, IGF-1-induced phosphorylation of the apoptotic protein BAD was inhibited by the PDE3 inhibitor cilostamide. Activated PKB phosphorylated and activated rPDE3B in vitro. Apparently, PDE3B, not PDE4, is a target of PKB and activated PDE3B may regulate cAMP pools that modulate effects of PKB on thymidine incorporation and BAD phosphorylation in FDCP2 cells.

IT 67763-96-6, IGF-1

RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(cyclic nucleotide phosphodiesterase 3B as downstream target of protein kinase B is involved in regulation of effects of protein kinase B on thymidine incorporation in FDCP2 promyeloid cells)

REFERENCE COUNT: 85 THERE ARE 85 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 8 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:279284 HCAPLUS

TITLE: Nutritionally induced anovulation in beef heifers: ovarian and endocrine function during realimentation and resumption of ovulation

AUTHOR(S): Bossis, I.; Wettemann, R. P.; Welty, S. D.; Vizcarra, J.; Spicer, L. J.

CORPORATE SOURCE: Department of Animal Science, Oklahoma Agricultural Experiment Station, Stillwater, OK, 74078, USA

SOURCE: Biology of Reproduction (2000), 62(5), 1436-1444
CODEN: BIREBV; ISSN: 0006-3363

PUBLISHER: Society for the Study of Reproduction

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Nutritionally induced anovulatory and cyclic Angus .times. Hereford heifers were used to evaluate follicular growth and concns. of hormones and metabolites during anovulation and resumption of ovulation. Anovulatory heifers were fed to gain 0.6 (LGAIN) or 1.5 (HGAIN) kg/day until resumption of ovulation, and heifers with normal estrous cycles were fed a maintenance diet (M). Follicles .gtoreq. 4 mm in diam. were measured by daily ultrasonog. in HGAIN and LGAIN heifers during one follicular wave before realimentation (Wan) and in two waves (W-2, W-1) immediately before the wave resulting in first ovulation or luteinization (W0). Ovaries of M heifers were evaluated to det. the day of ovulation of the second-wave dominant follicle (DF). Resumption of ovulation after realimentation occurred 23 days earlier in HGAIN than in LGAIN. Maximum diam., growth rate, and persistence of dominant follicles increased, while persistence of first subordinate follicles decreased between anovulation and resumption of ovulation in anovulatory heifers. Concns. of LH in serum were similar for HGAIN and LGAIN and gradually increased during realimentation. The increase in estradiol before the first ovulation was less in realimented heifers compared with cyclic heifers. Concns. of insulin-like growth factor-I (IGF-I) in HGAIN and LGAIN gradually increased during realimentation but were lower than concns. of IGF-I in cyclic heifers at ovulation. Increased diam., growth rate, and persistence of the DF were assocd. with increased concns. of LH, estradiol, and IGF-I during the transition from nutritionally

induced anovulation to resumption of ovulatory cycles.

REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 9 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 2000:48670 HCAPLUS

DOCUMENT NUMBER: 132:217441

TITLE: Effect of Restricted Food Intake on Production, Catabolism, and Effects of **IGF-I**

and **Cyclic** Nucleotides in Cultured Ovarian Tissue of Domestic Nutria (*Myocastor coypus*)

AUTHOR(S): Sirotkin, A. V.; Martin, D.; Suvegova, K.; Makarevich, A. V.; Genieser, H.-G.; Luck, M. R.; Osadchuk, I. V.

CORPORATE SOURCE: Research Institute of Animal Production, Nitra, 949 92, Slovakia

SOURCE: General and Comparative Endocrinology (2000), 117(2), 207-217

CODEN: GCENA5; ISSN: 0016-6480

PUBLISHER: Academic Press

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The aims of these in vitro expts. were to examine the effects of short-term food restriction on ovarian secretory activity and the role of **IGF-I** and cAMP- and cGMP-dependent intracellular mechanisms in the control of ovarian function in domestic nutria. Slices of ovary from sexually mature animals kept under conditions of normal and restricted (1/2 of std. ratio) feeding were cultured with or without **IGF-I** (50 ng/mL), cAMP analogs (dbcAMP and Rp-cAMPS), and cGMP analogs (8-pCPT-cGMP and Rp-8-Br-PET-cGMPS; all at 100 nM). In nonovarian cells dbcAMP activates and Rp-cAMPS inhibits protein kinase A, while 8-p-CPT-cGMP activates and Rp-8-Br-PET-cGMPS inhibits protein kinase G and cGMP-gated ion channels. **IGF-I** release and catabolism, as well as the release of progesterone (P), estradiol (E), and cAMP by the cultures, were evaluated using RIA. **IGF-I** did not affect cAMP release, while each of the cAMP and cGMP analogs inhibited **IGF-I** release in both control and exptl. groups. Fasting did not affect cAMP or **IGF-I** release. It partially prevented the effect of Rp-cAMPS, but not of other **cyclic** nucleotides, on **IGF-I** release and inhibited **IGF-I** catabolism. The Rp-cAMPS and Rp-8-Br-PET-cGMPS also inhibited **IGF-I** catabolism and the effects were greater with tissue from food-restricted than control animals. Ovaries from the underfed nutria secreted significantly more P and less E than those from normally fed animals. **IGF-I** and both cAMP analogs, given alone, did not affect P release, whereas a combination of **IGF-I** and Rp-cAMPS increased P output in control, but not in the exptl. group. The 8-pCPT-cGMP had no effect on P release. Rp-8-Br-PET-cGMPS, given alone or in combination with **IGF-I**, dramatically increased P secretion by tissue from control but not underfed animals. Estradiol secretion by tissue from underfed animals was stimulated by **IGF-I**, dbcAMP, Rp-cAMPS, 8-pCPT-cGMP, and Rp-8-Br-PET-cGMPS as well as by combinations of **IGF-I** and Rp-cAMPS or Rp-8-Br-PET-cGMPS; these effects were not seen with control tissue. The results demonstrate that: ovaries of domestic nutria secrete **IGF-I**, P, E, and cAMP; cAMP and cGMP can influence **IGF-I** release and catabolism; the **cyclic** nucleotides may have an **IGF-I**-mediated effect on P and E output; **IGF-I** and **cyclic** nucleotides can prevent the effect of undernutrition on E, but not on P release; effects of cAMP and cGMP on P and E are probably not mediated by protein kinase A, protein kinase G, or cGMP-gated ion channels; and food restriction can influence ovarian **IGF-I** catabolism, P, and E release and modulate the effects of **cyclic** nucleotides and **IGF-I** on steroidogenesis. It is concluded that ovarian secretory activity may be regulated sep. by nutrition and the **cyclic** nucleotide-**IGF-I** system, and there may be functional interrelationships between these mechanisms. (c) 2000 Academic Press.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 10 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:274432 HCAPLUS

DOCUMENT NUMBER: 131:57250

TITLE: Cyclic stretch regulates autocrine IGF-I in vascular smooth muscle cells: implications in vascular hyperplasia

AUTHOR(S): Standley, Paul R.; Obards, Tamar J.; Martina, Cherie L.

CORPORATE SOURCE: Department of Physiology, Midwestern University, Glendale, AZ, 85308, USA

SOURCE: American Journal of Physiology (1999), 276(4, Pt. 1), E697-E705

CODEN: AJPHAP; ISSN: 0002-9513

PUBLISHER: American Physiological Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Vascular smooth muscle cells (VSMC) subjected to acute or chronic stretch display enhanced growth rates in vitro and in vivo. Clin. examples of vascular hyperplasia (e.g., systolic hypertension and postinjury restenosis) suggest that local IGF-I expression is enhanced. Therefore, we investigated the role of in vitro cyclic stretch on rat VSMC IGF-I secretion and cellular growth. In serum-free medium, cyclic stretch (1 Hz at 120% resting length for 48 h) stimulated thymidine incorporation ~40% above that seen in nonstretched cells. Graded stretch magnitude (100-125% resting length) yielded graded increases in VSMC growth. Exogenous IGF-I increased growth of serum-starved, nonstretched VSMC in a dose-dependent manner, with maximal growth seen with 10⁻⁷ M. IGF-I secretion from stretched cells was 20- to 30-fold greater than from those cells cultured in a static environment. Stretch-induced increases in growth were completely blocked on addn. of anti-IGF-I and partially blocked with platelet-derived growth factor (PDGF) antibodies and with a tyrosine kinase inhibitor (tyrphostin-1). Finally, blockade of stretch-activated cation channels with GdCl₃ profoundly inhibited stretch-induced growth. We conclude that stretch increases VSMC IGF-I secretion and that such autocrine IGF-I is required for stretch-induced growth. PDGF and stretch-sensitive cation channels are likely, addnl. components of a complex pathway that regulates stretch-induced VSMC seen in systolic hypertension and postinjury restenosis.

IT 67763-96-6, IGF-I

RL: ADV (Adverse effect, including toxicity); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)

(cyclic stretch regulates autocrine IGF-I in vascular smooth muscle cells and implications in vascular hyperplasia)

REFERENCE COUNT: 58 THERE ARE 58 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 11 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:214466 HCAPLUS

DOCUMENT NUMBER: 131:39875

TITLE: Modification of plasma insulin-like growth factors and binding proteins during oral contraceptive use and the normal menstrual cycle

AUTHOR(S): Westwood, Melissa; Gibson, J. Martin; Pennells, Louise A.; White, Anne

CORPORATE SOURCE: Endocrine Sciences Research Group, Department of Medicine, and the School of Biological Sciences, University of Manchester, Manchester, UK

SOURCE: American Journal of Obstetrics and Gynecology (1999), 180(3, Pt. 1), 530-536

PUBLISHER: CODEN: AJOGAH; ISSN: 0002-9378
 Mosby, Inc.
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB Sex steroid regulation of the insulin-like growth factor axis is a subject of contention. The authors examd. the effect of combined oral contraceptives and investigated the cyclic variations in the insulin-like growth factor axis. Fasting blood samples were taken from 9 women receiving oral contraceptives, 10 women receiving no medication, and 10 male subjects. In women receiving oral contraceptives, insulin-like growth factor binding protein 1 remained highly phosphorylated and levels were acutely increased by sex steroid treatment (305 .mu.g/L on day 14 of the cycle [medication phase] vs. 118 .mu.g/L during the medication-free period). In women receiving no medication, insulin-like growth factor binding protein 1 levels were significantly lower (69 .mu.g/L on day 14 of the menstrual cycle) and varied cyclically, with a rise in the late-secretory phase that coincided with the appearance of nonphosphorylated and less phosphorylated insulin-like growth factor binding protein 1 isoforms. Compared with those in untreated women and in men, insulin-like growth factor I levels were decreased in women receiving oral contraceptives (405 ng/mL in untreated women and 330 ng/mL in men vs. 287 ng/mL in women receiving oral contraceptives). Oral contraceptive use had no effect on insulin-like growth factor II levels, and neither insulin-like growth factor I nor **insulin-like growth factor II** showed **cyclic** variation. The bioavailability of insulin-like growth factor I is reduced in users of oral contraceptives. This may contribute to the metabolic changes obsd. in such subjects.

REFERENCE COUNT: 25 THERE ARE 25 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 12 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1999:133022 HCAPLUS

DOCUMENT NUMBER: 130:333074

TITLE: The effects of equine somatotropin (eSt) on follicular development and circulating plasma hormone profiles in cyclic mares treated during different stages of the estrous cycle

AUTHOR(S): Cochran, R. A.; Leonardi-Cattolica, A. A.; Sullivan, M. R.; Kincaid, L. A.; Leise, B. S.; Thompson, D. L., Jr.; Godke, R. A.

CORPORATE SOURCE: Department of Animal Science, LSU Agricultural Center, Louisiana State University, Baton Rouge, LA, 70803, USA

SOURCE: Domestic Animal Endocrinology (1999), 16(1), 57-67
 CODEN: DANEEE; ISSN: 0739-7240

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of exogenous equine somatotropin (eST) administration on ovarian activity and plasma hormone levels were evaluated on horse and pony mares. The objectives of this study were to det. the effects of eST on follicular development and circulating concns. of LH (LH), estradiol, progesterone, and **insulin-like growth factor I (IGF-I)** in **cyclic** horse and pony mares. Sixteen mares received daily injections (i.m.) of eST at a concn. of 25 .mu.g/kg body wt. on either Days 6 through 12 (Treatment A) or 13 through 19 (Treatment B) postovulation. In addn., contemporary mares were similarly given the carrier vehicle and served as controls (Treatments C and D). Blood samples were collected at 24-h intervals and ultrasonog. evaluations were performed on the ovaries of each mare at 48-h intervals beginning on the first day of treatment and ending either on the day of ovulation or 5 d postovulation. Circulating

levels of insulin-like growth factor-I (IGF-I) were increased in treated mares by Day 3 post-treatment ($P < 0.05$). Also, mares in Treatment B exhibited a decrease in plasma estradiol concns. ($P < 0.05$) when compared with control mares on Days 1 through 5 postovulation of the post-treated estrous cycle. In addn., circulating LH levels were different for mares in Treatment A compared with controls on Days -8 through -1 pre-ovulation ($P < 0.05$). All follicles present on the ovaries of each mare were measured and placed into one of five categories based on their diam. Neither the mean no. of follicles per size category ≥ 8 mm in diam. nor the mean follicular diam. within each size category differed among treatment and control mares. However, eST treatment significantly increased the no. of follicles ≥ 7 mm on the ovaries of mares treated early in the estrous cycle when compared with control mares on Days 3 and 7 post-treatment and at the onset of standing estrus.

REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 13 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:597963 HCAPLUS

DOCUMENT NUMBER: 130:14243

TITLE: New intrachain cyclic nonapeptides of human IGF I and IGF II: synthesis and some properties

AUTHOR(S): Velek, J.; Barth, T.; Cerna, B.; Hauzerova, L.; Jiracek, J.; Pacakova, V.; Skarda, J.; Jeek, J.; Barthova, J.; Ubik, K.

CORPORATE SOURCE: Inst. of Organic Chemistry and Biochemistry, Academy of Sciences, Prague, 166 10/6, Czech Rep.

SOURCE: Peptides 1996, Proceedings of the European Peptide Symposium, 24th, Edinburgh, Sept. 8-13, 1996 (1998), Meeting Date 1996, 869-870. Editor(s): Ramage, Robert; Epton, Roger. Mayflower Scientific: Kingswinford, UK.

CODEN: 66RCA5

DOCUMENT TYPE: Conference

LANGUAGE: English

AB A symposium report on the prepn. and characterization of human insulin-like growth factor I (IGF I) and IGF II cyclic peptide analogs H-Cys-Ala-X-Arg-Ser-Cys-Asp-Leu-Y cyclic disulfides (X = Phe, Y = Arg-NH₂, Arg-OH, Ala-NH₂, Ala-OH; X = Tyr, Y = Ala-NH₂, Arg-NH₂).

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L6 ANSWER 14 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1998:48532 HCAPLUS

DOCUMENT NUMBER: 128:162625

TITLE: The effects of tamoxifen on endometrial insulin-like growth factor-1 expression

AUTHOR(S): Elkas, John; Gray, Karen; Howard, Leonard; Petit, Nancy; Pohl, Joseph; Armstrong, Alicia

CORPORATE SOURCE: Divisions of Obstetrics and Gynecol., Natl. Naval Med. Cent., Bethesda, MD, USA

SOURCE: Obstetrics and Gynecology (New York) (1998), 91(1), 45-50

CODEN: OBGNAS; ISSN: 0029-7844

PUBLISHER: Elsevier Science Inc.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB This study examd. whether modulation of insulin-like growth factor-1 and insulin-like growth factor-binding protein-1 expression underlies the uterotrophic effects assocd. with tamoxifen therapy in postmenopausal breast cancer patients. Using immunohistochem. techniques, we analyzed 37 endometrial specimens from biopsies or hysterectomies for Ki-67,

insulin-like growth factor-1, and insulin-like growth factor-binding protein-1 expression. Specifically, five secretory- and three proliferative-phase endometrial specimens were used as controls; 20 specimens (including two endometrial adenocarcinomas) were analyzed from postmenopausal breast cancer patients treated with tamoxifen (20 mg/day) for at least 6 mo; and nine endometrial adenocarcinoma specimens from patients not treated with tamoxifen were studied. Intensity of immunostaining was quantified using digitized imaging techniques. Results: Insulin-like growth factor-1 and insulin-like growth factor-1-binding protein-1 were expressed in normal and neoplastic endometrium of all patients, regardless of tamoxifen treatment. However, **insulin-like growth factor-1** expression varied **cyclically** in histol. normal endometrium, was reduced in undifferentiated endometrial tumors, and was upregulated in tamoxifen-treated specimens. Insulin-like growth factor-binding protein-1 immunostaining did not vary during the menstrual cycle, but it was reduced significantly in benign tamoxifen-exposed tissue and endometrial adenocarcinomas, regardless of degree of differentiation or tamoxifen exposure. No correlation was found between the expression of insulin-like growth factor-1 and insulin-like growth factor-binding protein-1 and the proliferative indexes of the tissues examd. Conclusion: The expression of insulin-like growth factor-1 and insulin-like growth factor-binding protein-1 in the uterus supports an autocrine and/or paracrine role for these proteins in endometrial physiolo. Although further studies are needed, our investigation suggests that altered expression of insulin-like growth factor-1 and insulin-like growth factor-binding protein-1 may contribute to the uterotrophic effects of tamoxifen.

L6 ANSWER 15 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:717694 HCAPLUS

DOCUMENT NUMBER: 126:43084

TITLE: Insulin-like growth factors and their binding proteins in the ovine oviduct during the estrous cycle

AUTHOR(S): Stevenson, K. R.; Wathes, D. C.

CORPORATE SOURCE: Dep. Farm Animal Equine Medicine Surgery, Royal Veterinary College, Potters Bar, Herts, EN6 1NB, UK

SOURCE: Journal of Reproduction and Fertility (1996), 108(1), 31-40

CODEN: JRPFA4; ISSN: 0022-4251

PUBLISHER: Journals of Reproduction and Fertility Ltd.

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The oviduct is the site of fertilization, and factors present in the oviductal fluid appear to be crucial to the future success of conceptus development. The spatial and temporal localization of mRNA encoding components of the insulin-like growth factor (IGF) system (IGF-I, IGF-II, they type 1 IGF receptor, and IGF-binding proteins -2, -3 and -4) in the ovine oviduct were examd. in tissue samples taken during the early and late stages of follicular development, and the early, mid-, and late luteal phases using in situ hybridization. Expression of mRNA encoding **IGF-I** showed a **cyclical** pattern, increasing sharply in the mucosa and muscularis during the late follicular phase, then declining. In the muscularis, mRNA encoding IGF-II exhibited no temporal changes, but concns. in the mucosa increased from the late follicular stage to the early luteal phase. mRNA encoding the type 1 IGF receptor was present throughout the oviduct. Concns. increased during the follicular phase to peak in the early luteal phase in both the mucosa and muscularis. IGFBP-2 gene transcripts were undetectable at all time points examd. MRNAs encoding IGFBP-3 and IGFBP-4 were localized primarily in the stromal region. IGFBP-3 expression peaked in the late follicular stage of the cycle. The concn. of mRNA encoding IGFBP-4 increased in the follicular phase and was maintained at a significantly higher concn. during the early and mid-luteal stages. The coordinate max. expression of

mRNA for both IGF-I and IGF-II, the type 1 IGF receptor and IGFBP-3 during the period when the gametes and embryo are in transit suggests a role for IGF-I and IGF-II peptides in providing an oviductal environment propitious to conception and early embryonic growth and metab.

L6 ANSWER 16 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:344455 HCAPLUS
DOCUMENT NUMBER: 125:26469
TITLE: Native and non-native structure in a protein-folding intermediate: spectroscopic studies of partially reduced IGF-I and an engineered alanine model
AUTHOR(S): Hua, Qing-Xin; Narhi, Linda; Jia, Wenhua; Arakawa, Tsutomu; Rosenfeld, Robert; Hawkins, Nessa; Miller, James A.; Weiss, Michael A.
CORPORATE SOURCE: Cent. Biomol. Struct. Function, Univ. Chicago, Chicago, IL, 60637, USA
SOURCE: Journal of Molecular Biology (1996), 259(2), 297-313
CODEN: JMOBAK; ISSN: 0022-2836
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The structure of a metastable folding intermediate of human insulin-like growth factor I (IGF-I) and an engineered model are investigated by CD and two-dimensional 1H NMR spectroscopy. The intermediate, which contains two of three native disulfide bonds, was trapped by acid quenching and isolated by reverse-phase HPLC. The reduced cysteine residues were mapped to residues 47 and 52 (corresponding to A6-A11 in insulin). In the native state this disulfide bridge anchors an adjoining amphipathic .alpha.-helix (helix 2; residues 42 to 49) against the hydrophobic core. Comparison of CD and 1H-NMR spectra demonstrates that the acid-quenched intermediate is partially folded and contains elements of native secondary and tertiary structure. Spectra are similar to those of an equil. model in which the reduced cysteine residues are replaced by alanine. Complete 1H-NMR sequential assignment of the alanine model has been obtained and demonstrates that removal of the disulfide bond is assocd. with local unfolding of the adjoining .alpha.-helix. Native secondary structure (including helices 1 and 3) is otherwise retained and defines a folded subdomain. Long-range nuclear Overhauser effects (NOE) within this subdomain are similar to those of native IGF-I; no non-native NOE is obsd. Our results support the hypothesis that folding of the insulin motif is directed by a subset of native structural elements and that these elements form at an early step in the pathway. Formation of helix 2, despite its prominence in the native state, is likely to represent a late step. Hydrophobic collapse of this segment appears to precede helix formation.

IT 122635-51-2, **Cyclic** (6.fwdarw.47), (18.fwdarw.61), (48.fwd

arw.52)-tris(disulfide) human IGF-I

RL: PRP (Properties)

(spectroscopic studies of partially reduced IGF-I and an engineered alanine model in relation to native and non-native structure in a protein-folding intermediate)

L6 ANSWER 17 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1996:47615 HCAPLUS
DOCUMENT NUMBER: 124:166079
TITLE: IRS-I expression on the luteinized rat ovary: IGF-I and **cyclic** AMP effects on IRS-I tyrosine phosphorylation
AUTHOR(S): Talavera, Francisco; Chen, Zhouji; Menon, K. M. J.
CORPORATE SOURCE: Ann Arbor, MI, 48109-0278, USA
SOURCE: Biochimica et Biophysica Acta (1996), 1310(1), 10-18
CODEN: BBACAQ; ISSN: 0006-3062
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal

LANGUAGE: English

AB The expression of insulin receptor substrate-I (IRS-I) mRNA was demonstrated in rat luteal cells by Northern blot anal., in situ hybridization as well as by reverse transcriptase polymerase chain reaction. Western blot with a polyclonal anti IRS-I antibody showed the presence of a 183 kDa protein which corresponds to the size of IRS-I reported in other tissues. Further studies were performed to det. whether human chorionic gonadotropin (hCG) can interact with the insulin-like growth factor-I (IGF-I) signaling pathway to increase tyrosine phosphorylation of IRS-I. While hCG alone was ineffective in stimulating the phosphorylation of IRS-I, IGF-I mediated phosphorylation of IRS-I was increased by prior exposure to hCG. These results were further confirmed by the immunopptn. of IRS-I from the lysate of hCG- and IGF-I-treated luteal cell cultures followed by Western blotting with anti-phosphotyrosine antibody. Similarly, pretreatment with forskolin also increased IGF-I stimulated IRS-I phosphorylation. The increased tyrosine phosphorylation of IRS-I seen in response to IGF-I stimulation following treatment with either hCG or forskolin was not due to an increase in IRS-I content. Furthermore, IGF-I receptor tyrosine kinase activity was not affected by forskolin, suggesting that the increase in IRS-I tyrosine phosphorylation was not the result of an increase in its activity. Thus, the authors conclude that hCG/LH and IGF-I signaling pathways 'cross-talk' to increase the levels of IRS-I tyrosine phosphorylation. The obsd. increase in IRS-I tyrosine phosphorylation may be the result of an increase in the stability of the phosphorylated form of IRS-I.

L6 ANSWER 18 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:833975 HCAPLUS

DOCUMENT NUMBER: 123:223765

TITLE: Tissue specific and **cyclic** expression of **insulin-like growth factor** binding proteins -1, -2, -3, -4, -5, -6 in the rat oviduct

AUTHOR(S): Erickson, Gregory F.; Grivigian, Michael R.; Sadighian, Ali R.; Nakatani, Akira; Ling, Nicholas; Shimasaki, Shunichi

CORPORATE SOURCE: Dep. of Reproductive Medicine, Univ. of California, San Diego, CA, 92093-0674, USA

SOURCE: Endocrine (1995), 3(9), 667-76
CODEN: EOCRE5; ISSN: 1355-008X

PUBLISHER: Macmillan Scientific & Medical Division

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Although much is known about the expression insulin-like growth factors (IGF) and their receptors in the murine oviduct, significantly less is known about the expression of IGF binding proteins (IGFBPs). To fill this gap in the knowledge, the authors identified and characterized the tissue specific expression of IGFBP-1 to -6 in rat oviducts over the estrous cycle by in situ hybridization and immunocytochem. Tissues were analyzed on proestrus (P1000 h, P2000 h), estrus (E0200, E1000 h), and diestrus I and II (DI 1100 h, DII 1100 h). IGFBP-1 was undetectable in the oviduct over the cycle. IGFBP-2 was selectively expressed in the luminal epithelium. The mRNA levels were high between P2000 h and E1000 h but low or undetectable thereafter. Immunoreactive IGFBP-2 was strong to very strong in these cells over most of the cycle. IGFBP-3 mRNA was undetectable in the oviduct; however, strong hybridization and immunoreactive signals were present in the mesosalpinx and mesotubarium, particularly at DI and DII. IGFBP-4 mRNA was not detected in the oviduct; however, strong hybridization and immunoreactive signals were present in the mesosalpinx and mesotubarium, particularly at DI and DII. IGFBP-4 mRNA was not detected in the oviduct. In contrast, immunoreactive IGFBP-4 was obsd. in the luminal epithelium and the intensity was very strong

after ovulation (E1000 h, DI and DII). IGFBP-5 and -6 mRNAs were selectively expressed in circular smooth muscle cells. Hybridization signals were evident over the cycle, but were greatest at estrus. By comparison, IGFBP-5 and -6 proteins were essentially undetectable in these cells except at DII 1100 h when immunostaining was moderate to high. Luminal epithelial cells were weakly pos. for IGFBP-5 and -6. However, intense immunostaining was assocd. with the ciliated border and the luminal fluid juxtaposed to these cells during the cycle. The oocyte-cumulus complexes were immunostained intensely for IGFBP-2, -4, -5 and -6, but their mRNAs were undetectable. The signals were strongest in degenerating cumulus cells suggesting a potential role for these IGFBPs in cumulus apoptosis. These results demonstrate that the estrous cycle is accompanied by major changes in the pattern of expression of IGFBP-2, -4, -5 and -6 in the rat oviduct. The authors therefore conclude that the regulated prodn. of these particular IGFBPs may be functionally important in modulating IGF activities in the oviduct, oocyte cumulus complexes, and perhaps the preimplantation embryo as well.

L6 ANSWER 19 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:761354 HCAPLUS

DOCUMENT NUMBER: 123:161681

TITLE: Regulation of **insulin-like growth factor I** transcription by **cyclic** adenosine 3',5'-monophosphate (cAMP) in fetal rat bone cells through an element within exon 1: protein kinase a-dependent control without a consensus cAMP response element

AUTHOR(S): McCarthy, Thomas L.; Thomas, Michael J.; Centrella, Michael; Rotwein, Peter

CORPORATE SOURCE: Sectino of Plastic Surgery, Yale Univ. Sch. Medicine, New Haven, CT, 06520-8041, USA

SOURCE: Endocrinology (1995), 136(9), 3901-8
CODEN: ENDOAO; ISSN: 0013-7227

PUBLISHER: Endocrine Society

DOCUMENT TYPE: Journal

LANGUAGE: English

AB Insulin-like growth factor I (IGF-I) is a locally synthesized anabolic growth factor for bone. IGF-I synthesis by primary fetal rat osteoblasts (Ob) is stimulated by agents that increase the intracellular cAMP concn., including prostaglandin E2 (PGE2). Previous studies with Ob cultures demonstrated that PGE2 enhanced IGF-I transcription through selective use of IGF-I promoter 1, with little effect on IGF-I mRNA half-life. Transient transfection of Ob cultures with an array of promoter 1-luciferase reporter fusion constructs has now allowed localization of a potential cis-acting promoter element(s) responsible for cAMP-stimulated gene expression to the 5'-untranslated region (5'-UTR) of IGF-I exon 1, within a segment lacking a consensus cAMP response element. This evidence derives from three principal observations: (1) a transfection construct contg. only 122 nucleotides (nt) of promoter 1 and 328 nt of the 5'-UTR retained full PGE2-stimulated reporter expression; (2) maximal PGE2-driven reporter expression required the presence of nt 196 to 328 of exon 1 when tested within the context of IGF-I promoter 1; (3) cotransfection of IGF-I promoter-luciferase-reporter constructs with a plasmid encoding the .alpha.-isoform of the catalytic subunit of murine cAMP-dependent protein kinase (PKA) produced results comparable to those seen with PGE2 treatment, whereas cotransfection with a plasmid encoding a mutant regulatory subunit of PKA that cannot bind cAMP blocked PGE2-induced reporter expression. DNase I footprinting of the 5'-UTR of exon 1 demonstrated protected sequences at HS3A, HS3B, and HS3D, three of six DNAProtein binding sites previously characterized with rat liver nuclear exts. Of these three regions, only the HS3D binding site is located within the functionally identified hormonally responsive segment of IGF-I

exon 1. These results directly implicate PKA in the control of IGF-I gene transcription by PGE2 and identify a segment of IGF-I exon 1 as being essential for this hormonal regulation.

L6 ANSWER 20 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:740211 HCAPLUS
DOCUMENT NUMBER: 123:133617
TITLE: Divergence in macrophage insulin-like growth factor-I (IGF-I) synthesis induced by TNF-.alpha. and prostaglandin E2
AUTHOR(S): Fournier, Thierry; Riches, David W. H.; Winston, Brent W.; Rose, David M.; Young, Scott K.; Noble, Paul W.; Lake, Fiona R.; Henson, Peter M.
CORPORATE SOURCE: Dep. Pediatrics, Natl. Jewish Cent. Immunol. Respiratory Med., Denver, CO, 80206, USA
SOURCE: Journal of Immunology (1995), 155(4), 2123-33
CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER: American Association of Immunologists
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Increased synthesis of insulin-like growth factor I (IGF-I), a fibroblast growth factor, is induced in murine macrophages by TNF-.alpha.. TNF-.alpha. also induces macrophages to express cytotoxic activity, but only during costimulation with interferons. Since prostaglandin E2 (PGE2) is known to inhibit macrophage cytotoxic activity, its possible reciprocal enhancement of IGF-I synthesis was examined. PGE2 or dibutyryl cAMP (dbcAMP) stimulated the synthesis of IGF-I similarly to TNF-.alpha. in magnitude and time course. TNF-.alpha. did not increase IGF-I synthesis by first inducing PGE2 synthesis, because indomethacin was unable to block the effect of TNF-.alpha.. PGE2 did not stimulate IGF-I synthesis by first inducing TNF-.alpha. production, because (1) anti-TNF-.alpha. antibody (Ab) did not block PGE2-induced IGF-I synthesis, and (2) PGE2 down-regulated TNF-.alpha. mRNA levels and did not affect levels of the cytokine in supernatants. Moreover, the difference in the induction of IGF-I was observed at the level of signal transduction, in that PGE2 and dbcAMP increased cAMP-dependent protein kinase (PKA) activity, whereas TNF-.alpha. stimulated the mitogen-activated protein (MAP) kinase pathway. Divergence between the two pathways was also noted in the regulation of IGF-I at the mRNA level, and an additive effect on IGF-I synthesis was observed when cells were incubated with the combination of TNF-.alpha. plus PGE2 or dbcAMP. Collectively, these data suggest that TNF-.alpha. and PGE2 stimulate IGF-I synthesis in macrophages by two separate pathways, and that PGE2 acts as a positive stimulus for IGF-I synthesis through a cAMP/PKA pathway.

L6 ANSWER 21 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1995:356265 HCAPLUS
DOCUMENT NUMBER: 122:129322
TITLE: Regulation of expression of surface aminopeptidase N in human glomerular mesangial cells. II. Effect of cyclic nucleotides, growth factors and mitogens
AUTHOR(S): Stefanovic, Vladislav; Vlahovic, Predrag
CORPORATE SOURCE: Inst. of Nephrology and Hemodialysis, Fac. of Medicine, Nis, Yugoslavia
SOURCE: Cellular Physiology and Biochemistry (1995), 5(2), 127-34
CODEN: CEPBEW; ISSN: 1015-8987
PUBLISHER: Karger
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Aminopeptidase N of human glomerular mesangial cells is an ectoenzyme. Control of its expression by mitogens has been demonstrated. Aminopeptidase N activity was increased after 72 h of treatment with

8-bromo-cAMP and two cAMP-stimulating agents, forskolin and isobutylmethylxanthine (IBMX). IBMX (100 μ M), a phosphodiesterase inhibitor, stimulated aminopeptidase N activity by 40%. Stimulation by cAMP and forskolin was less marked, 25 and 28%, resp. Phorbolmyristate acetate (PMA) treatment increased enzyme activity by 80%; however, after treatment with both PMA and cAMP or PMA and forskolin, aminopeptidase N activity increased by 178 and 190%, resp. Bacterial lipopolysaccharide (LPS) moderately stimulated surface aminopeptidase N activity; LPS potentiated the effect of PMA on enzyme activity. Thrombin (0.1-5 U/mL) also stimulated aminopeptidase N activity, by 27% at a concn. of 2.5 U/mL. The effects of thrombin and cAMP for forskolin were additive. Dexamethasone (0.1-10 μ M) treatment for up to 6 days was without effect on aminopeptidase N expression. A short treatment (72 h) with interferon- γ (1-5,000 U/mL) increased enzyme activity by 26%. The effect of insulin growth factor I (50 ng/mL) and endothelin (0.1-100 nM) on aminopeptidase N activity was not significant. Serum withdrawal from the culture medium was accompanied by a significant increase in enzyme activity in the 48-h culture. This study shows that expression of mesangial cell aminopeptidase N is regulated by mitogens, cAMP and c-AMP-stimulating agents acting in concert.

L6 ANSWER 22 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:672355 HCAPLUS

DOCUMENT NUMBER: 121:272355

TITLE: Similarities in the regulation of hIGFBP-1 and PEPCK gene expression

AUTHOR(S): Powell, David R.; Lee, Phillip D. K.; Suwanichkul, Adisak

CORPORATE SOURCE: Department Pediatrics, Baylor College Medicine, Houston, TX, 77030, USA

SOURCE: International Congress Series (1994), 1056(INSULIN-LIKE GROWTH FACTORS AND THEIR REGULATORY PROTEINS), 141-50
CODEN: EXMDA4; ISSN: 0531-5131

DOCUMENT TYPE: Journal; General Review

LANGUAGE: English

AB A review, with 48 refs., on regulation of human insulin-like growth factor binding protein-1 (hIGFBP-1) expression is similar to that of phosphoenolpyruvate carboxykinase (PEPCK), a key enzyme in gluconeogenesis. Both genes are expressed primarily in liver and kidney. As with PEPCK, hepatic expression of hIGFBP-1 is regulated by multiple hormones (e.g., insulin and corticosteroids) and cAMP and primarily at the level of transcription and many cis elements important to this regulation are located in the first 460 base pairs (bp) 5' to the transcription start site. Comparison of their organization and function suggests that the PEPCK and hIGFBP-1 promoters use similar cis elements and trans-acting factors in a different spatial organization to achieve similar effects.

L6 ANSWER 23 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1994:549378 HCAPLUS

DOCUMENT NUMBER: 121:149378

TITLE: Interrelationships among estrogen, **insulin-like growth factor-I** and **cyclic** adenosine monophosphate in the regulation of uterine progesterone and estrogen receptors

AUTHOR(S): Aronica, Susan Marie

CORPORATE SOURCE: University of Illinois, Urbana, IL, USA

SOURCE: (1994) 173 pp. Avail.: Univ. Microfilms Int., Order No. DA9416335
From: Diss. Abstr. Int. B 1994, 55(1), 52-3

DOCUMENT TYPE: Dissertation

LANGUAGE: English

AB Unavailable

L6 ANSWER 24 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1993:464793 HCAPLUS

DOCUMENT NUMBER: 119:64793

TITLE: Identification of a promoter element which participates in cAMP-stimulated expression of human insulin-like growth factor-binding protein-1

AUTHOR(S): Suwanichkul, Adisak; DePaolis, Laura A.; Lee, Phillip D. K.; Powell, David R.

CORPORATE SOURCE: Dep. Pediatr., Baylor Coll. Med., Houston, TX, 77030, USA

SOURCE: Journal of Biological Chemistry (1993), 268(13), 9730-6

CODEN: JBCHA3; ISSN: 0021-9258

DOCUMENT TYPE: Journal

LANGUAGE: English

AB HEP G2 cells were used to evaluate the role of cAMP in stimulating insulin-like growth factor-binding protein -1 (IGFBP-1) expression. Initial studies found that either 0.5 or 5.0 mM dibutyryl cAMP (Bt2cAMP) alone, or the combination of 0.5 mM Bt2cAMP and 2 mM theophylline, increased IGFBP-1 protein levels, mRNA levels, and promoter activity, but that the addn. of theophylline to Bt2cAMP was required to give a .apprx.5-fold increase in promoter activity. Deletion mutations of the IGFBP-1 promoter were used to show that much of the effect of Bt2cAMP and theophylline was conferred by the region between 269 and 246 base pairs (bp) 5' of the IGFBP-1 mRNA cap site. DNase I protection assays showed that HEP G2 nuclear ext. footprinted the region from 273 to 249 bp 5' of the cap site; this region, designated P2, has a central CGTCA motif common to cAMP-responsive elements (CREs). Mutating the CGTCA motif in the 1205-bp IGFBP-1 promoter construct to TAGCA led to a 51% decrease in the ability of Bt2cAMP and theophylline to stimulate IGFBP-1 promoter activity above control levels. In addn., cotransfection of the catalytic subunit of cAMP-dependent protein kinase A (PKA) with the native 1205-bp IGFBP-1 promoter construct stimulated IGFBP-1 promoter activity 3.9-fold, but the TAGCA mutation decreased by 73% the ability of PKA to stimulate IGFBP-1 promoter activity above control levels. Mutating the CGTCA motif to TAGCA also blocked the ability of both crude HEP G2 nuclear ext. and recombinant CRE-binding protein to bind to the P2 element. These data suggest that the P2 element is a CRE that confers at least part of the stimulatory effect of cAMP on the human IGFBP-1 promoter.

L6 ANSWER 25 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1992:35054 HCAPLUS

DOCUMENT NUMBER: 116:35054

TITLE: Human chorionic gonadotropin up-regulates insulin-like growth factor-I receptor gene expression of Leydig cells

AUTHOR(S): Nagpal, Madan L.; Wang, Deli; Calkins, Jo H.; Chang, Weiwei; Lin, Tu

CORPORATE SOURCE: Med. Serv., W. J. B. Dorn Veterans Hosp., Columbia, SC, 29201, USA

SOURCE: Endocrinology (1991), 129(6), 2820-6

CODEN: ENDOAO; ISSN: 0013-7227

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The effects of human chorionic gonadotropin (hCG) 8-bromo-cAMP, PMA, and forskolin on IGF-I receptor gene expression of Leydig cells were studied. The treatment of purified Leydig cells with hCG caused a dose-dependent increase in [125I]IGF-I binding to Leydig cells without changes in binding affinity, indicating that the increased binding was due to increased receptor nos. and not to increased affinity. The minimal time required for hCG to induce IGF-I binding was 6 h, and it reached a plateau at 16 h.

8-Bromo-cAMP (1 mM) increased IGF-I binding about 2-fold, and forskolin (10 μ M) increased binding about 51%. The RNase protection assay showed that hCG and 8-bromo-cAMP increased IGF-I receptor mRNA expression as early as 2 h before the increase in IGF-I binding. The induction by hCG was >3.5-fold at 4 h and decreased to about 2-fold at 6 h. PMA had a very small effect on IGF-I receptor mRNA levels (1.5-fold increase at 2 h and no changes at 4 and 6 h). Thus, IGF-I receptors can be upregulated by hCG, 8-bromo-cAMP, and forskolin. The up-regulation of IGF-I receptor no. is assocd. with transient increases in IGF-I receptor mRNA levels. This could be a mechanism by which hCG and IGF-I interact to enhance Leydig cell steroidogenesis.

L6 ANSWER 26 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1990:585365 HCAPLUS
 DOCUMENT NUMBER: 113:185365
 TITLE: Regulation of 11.beta.- and 17.alpha.-hydroxylases in cultured bovine adrenocortical cells: 3',5'-cyclic adenosine monophosphate, insulin-like growth factor-I, and activators of protein kinase C
 AUTHOR(S): Naseeruddin, Syed A.; Hornsby, Peter J.
 CORPORATE SOURCE: Dep. Cell Mol. Biol., Med. Coll. Georgia, Augusta, GA, 30912, USA
 SOURCE: Endocrinology (1990), 127(4), 1673-81
 CODEN: ENDOAO; ISSN: 0013-7227
 DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The induction of steroid 11.beta.-hydroxylase and 17.alpha.-hydroxylase was studied in bovine adrenocortical cell cultures in serum-free medium. In the absence of insulin-like growth factor (IGF)-I or insulin, cholera toxin failed to increase 11.beta.-hydroxylase enzyme activity or mRNA levels; cholera toxin increased 11.beta.-hydroxylase activity and mRNA only in the presence of 10 nM IGF-I or of higher concns. of insulin. 17.alpha.-Hydroxylase enzyme activity and mRNA, in contrast, were increased maximally by cholera toxin in the absence of insulin or IGF. When cultures were incubated with cholera toxin, cAMP analogs, forskolin, ACTH, or PGE1 in defined medium with insulin, all agents increased the mRNA levels for 11.beta.-hydroxylase and 17.alpha.-hydroxylase. 11.beta.-Hydroxylase enzyme activity was detectable in control (insulin only) cultures and was increased to varying extents by the different agents. 17.alpha.-Hydroxylase enzyme activity was undetectable in control cultures and was increased more than 50-fold by all agents. The sensitivity of induction of 11.beta.-hydroxylase and 17.alpha.-hydroxylase enzyme activities by cAMP was compared by using serial dilns. of an equimolar mixt. of N6-monobutyl-8-bromo-cAMP and 8-bromo-cAMP. For both enzymes, the response curve was biphasic, with a maximal response in the range of 20 to 100 μ M each analog, but the decline in response at higher cAMP concns. was much more marked for 11.beta.-hydroxylase than for 17.alpha.-hydroxylase. The effects of activation of protein kinase C were studied in cultures incubated with 12-O-tetradecanoylphorbol-13-acetate (TPA) together with a cAMP analog mixt. TPA decreased cAMP-induced 11.beta.-hydroxylase mRNA; TPA also decreased the induction of 17.alpha.-hydroxylase mRNA, as previously reported. TPA caused a dose-dependent decrease in cAMP-induced 11.beta.-hydroxylase enzyme activity. Angiotensin II at 0.1 to 10 μ M also decreased induction of 11.beta.-hydroxylase. Induction of 11.beta.-hydroxylase and 17.alpha.-hydroxylase is coordinately regulated by cAMP, protein kinase C, and IGF-I/insulin, but responses to these regulators differ in various respects between these two cytochrome P 450 enzymes.

L6 ANSWER 27 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1990:585324 HCAPLUS

DOCUMENT NUMBER: 113:185324
 TITLE: Adipose conversion of 3T3-L1 cells in a serum-free culture system depends on epidermal growth factor, **insulin-like growth factor I**, corticosterone, and **cyclic AMP**
 AUTHOR(S): Schmidt, Wilfried; Poell-Jordan, Gisela; Loeffler, Georg
 CORPORATE SOURCE: Dep. Biochem., Microbiol. Genet., Univ. Regensburg, Regensburg, D-8400, Germany
 SOURCE: Journal of Biological Chemistry (1990), 265(26), 15489-95
 CODEN: JBCHA3; ISSN: 0021-9258
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB A culture system for 3T3-L1 preadipocytes based on a serum-free chem. defined medium contg. fetuin, transferrin, and pantothenate is described. In this system, adipose conversion depends on the following conditions. In the presence of high insulin concns. (1 μ M), addn. of corticosterone together with IBMX) for not more than the 1st 4 days after confluence to the culture medium induces maximal adipose conversion within 12-14 days. IBMX may be replaced by forskolin or permeable analogs of cAMP, indicating that its effect is due to elevated cellular cAMP levels. At low insulin concns. (1 nM), adipose conversion is reduced. Growth hormone or insulin-like growth factor I together with EGF have to be present as a medium supplement together with corticosterone and IBMX to get maximal adipose conversion. The induction of adipose conversion by corticosterone and IBMX in the presence of either high insulin concns. or insulin-like growth factor I together with EGF is accompanied by postconfluent mitoses. Inhibitors of DNA replication markedly reduce adipose conversion. Fibroblast growth factor and platelet-derived growth factor, although acting as potent mitogens on 3T3-L1 cells, do not support adipose conversion induced by corticosterone and IBMX.

L6 ANSWER 28 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1990:565806 HCAPLUS
 DOCUMENT NUMBER: 113:165806
 TITLE: **Cyclic AMP induces insulin-like growth factor I synthesis in osteoblast-enriched cultures**
 AUTHOR(S): McCarthy, Thomas L.; Centrella, Michael; Canalis, Ernesto
 CORPORATE SOURCE: Med. Cent., Saint Francis Hosp., Hartford, CT, 06105, USA
 SOURCE: Journal of Biological Chemistry (1990), 265(26), 15353-6
 CODEN: JBCHA3; ISSN: 0021-9258
 DOCUMENT TYPE: Journal
 LANGUAGE: English
 AB Isobutylmethylxanthine, forskolin, and dibutyryl cAMP, agents that elevate intracellular cAMP levels by discrete mechanisms, also enhanced the steady state transcript and polypeptide level of IGF-I in rat osteoblast-enriched cultured. The calcium ionophore ionomycin and phorbol myristate acetate did not increase IGF-I synthesis. In contrast, none of the agents tested increased the steady state transcript or polypeptide levels for IGF-II. The rat IGF-I gene is >90 kilobases in length, and contains at least 3 promoter regions. The present data represent the first demonstration of cAMP mediated IGF-I gene regulation and indicate the potential for preferential promoter usage for modulating IGF-I gene expression in bone.

L6 ANSWER 29 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1989:568073 HCAPLUS
 DOCUMENT NUMBER: 111:168073

TITLE: Regulation of IGF-I receptors by corticotropin and angiotensin-II in cultured bovine adrenocortical cells
 AUTHOR(S): Louveau, Isabelle; Penhoat, Armelle; Saez, Jose M.
 CORPORATE SOURCE: Hop. Debrousse, Lyon, 69322, Fr.
 SOURCE: Biochemical and Biophysical Research Communications (1989), 163(1), 32-6
 CODEN: BBRCA9; ISSN: 0006-291X

DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The effects of angiotensin II (A-II) and ACTH on insulin-like growth factor-I (IGF-I) receptors of bovine adrenocortical cells were investigated. Pretreatment of cells for 48 h with ACTH or A-II induced, in a dose-dependent manner, an increase in [¹²⁵I]IGF-I binding (ED₅₀ .simeq. 10⁻¹¹M, V_{max} = 10⁻¹⁰M with ACTH; ED₅₀ .simeq. 3.10⁻⁹M, V_{max} = 10⁻⁷M with A-II). This resulted from an increase in the no. of binding sites without modification of the binding affinity. Pretreatment with 8-bromo-cAMP (10⁻³M), a phorbol ester (PMA 10⁻⁷M) + ionophore A 23187 (10⁻⁷M) produced a pos. regulation of IGF-I receptors. Glucocorticoids did not mediate the effect of A-II and ACTH on IGF-I receptors. Since previous studies have shown that IGF-I increases ACTH and A-II receptors the present data indicate the existence of a reciprocal pos. trophic effect between IGF-I and the 2 hormones on the regulation of their specific membrane-bound receptors.

L6 ANSWER 30 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1988:32671 HCAPLUS

DOCUMENT NUMBER: 108:32671

TITLE: Insulin-like growth factor I action on rat anterior pituitary cells: effects of intracellular messengers on growth hormone secretion and messenger ribonucleic acid levels

AUTHOR(S): Morita, Shigeki; Yamashita, Shunichi; Melmed, Shlomo
 CORPORATE SOURCE: Sch. Med., Univ. California, Los Angeles, CA, 90048, USA

SOURCE: Endocrinology (1987), 121(6), 2000-6
 CODEN: ENDOAO; ISSN: 0013-7227

DOCUMENT TYPE: Journal
 LANGUAGE: English

AB It was previously shown that insulin-like growth factor (IGF-I) suppresses basal and growth hormone-releasing hormone (GHRH)-induced growth hormone (GH) gene transcription. CAMP is a putative intracellular mediator of GHRH action. The mechanism of IGF-I action on the somatotroph with or without cAMP activators was thus examd. Primary rat pituitary cells growing in serum-free medium were treated with IGF-I. GH secretion was measured by RIA, and mRNA levels were measured by hybridization to [32P]cDNA for GH. 8-Bromo-cAMP (8-Br-cAMP; 0.625 mM) stimulated GH mRNA levels after 72 h by 238%. IGF-I (6.5 nM) caused a 64% inhibition of 8-Br-cAMP-stimulated GH mRNA levels and a similar inhibition of GH secretion. This inhibition was time and dose dependent, with maximal (71%) suppression of cAMP-induced GH achieved with 13 nM IGF-I after 72 h. Forskolin (1 .mu.M), a stimulator of adenylate cyclase, stimulated GH secretion (198%), which was inhibited by IGF-I by 42%. TPA, 50 nM, a potent activator of protein kinase C, strongly stimulated GH secretion (347%), which was similarly suppressed by IGF-I by 51%. The suppressive action of IGF-I on somatotroph gene expression is unimpaired by direct activation of both cAMP and protein kinase C, suggesting that IGF-I acts upon the GH gene by a mechanism that is not altered by these 2nd messengers. The neg. feedback inhibition of physiol. concns. of IGF-I on GH, therefore, appears to override the potent stimulation of GH by these intracellular messengers.

L6 ANSWER 31 OF 31 HCAPLUS COPYRIGHT 2003 ACS

ACCESSION NUMBER: 1982:504762 HCAPLUS

DOCUMENT NUMBER: 97:104762
 TITLE: Cyclic nucleotides and somatomedin action in cartilage
 AUTHOR(S): Stuart, Charles A.; Vesely, David L.; Provow, Sally
 A.; Furlanetto, Richard W.
 CORPORATE SOURCE: Med. Branch, Univ. Texas, Galveston, TX, 77550, USA
 SOURCE: Endocrinology (1982), 111(2), 553-8
 CODEN: ENDOAO; ISSN: 0013-7227

DOCUMENT TYPE: Journal
 LANGUAGE: English

AB The role of cyclic nucleotides was evaluated in the stimulation of cartilage metab. by somatomedin-C (Sm-C) [61461-67-4]. Tissue concns. of cAMP [60-92-4] in chick cartilage declined rapidly during organ culture, despite the presence of serum or Sm-C. The cGMP [7665-99-8] concns. in cartilage declined rapidly during control incubations, but were augmented significantly at 30 and 60 min of incubation with the addn. of serum or Sm-C. Thereafter, cGMP concns. declined toward the levels of incubated control cartilages. Sm-C had no effect on cAMP phosphodiesterase [9036-21-9] activity. N6-monobutyl cAMP [13117-60-7] Stimulated sulfate uptake, but dibutyl cGMP [32266-35-6] did not. Sm-C did not stimulate adenylate cyclase [9012-42-4] in purified plasma membranes from chondrocytes, whereas it stimulated both plasma membrane and cytosol guanylate cyclase [9054-75-5] at concns. of Sm-C as low as 10⁻¹²M. Thus, cAMP is not the intracellular 2nd messenger for Sm-C in cartilage, whereas cGMP may be a 2nd messenger mediating a portion of Sm's stimulation of cartilage metab.

IT **61461-67-4**

RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
 (cyclic nucleotides of cartilage response to)

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NEWS	23	Feb 24	TEMA now available on STN
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NEWS	30	Apr 11	Display formats in DGENE enhanced
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NEWS	32	Apr 17	Polymer searching in REGISTRY enhanced
NEWS	33	Apr 21	Indexing from 1947 to 1956 being added to records in CA/CAPLUS
NEWS	34	Apr 21	New current-awareness alert (SDI) frequency in WPIDS/WPINDEX/WPIX
NEWS	35	Apr 28	RDISCLOSURE now available on STN
NEWS	36	May 05	Pharmacokinetic information and systematic chemical names added to PHAR
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NEWS	38	May 15	Supporter information for ENCOMPAT and ENCOMPLIT updated
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E6	24	MERCAPTOPROPEN/BI
E7	5	MERCAPTOPROPENO/BI

E8	4	MERCAPTOPROPENOATE/BI
E9	1	MERCAPTOPROPENOATO/BI
E10	9	MERCAPTOPROPENOIC/BI
E11	15	MERCAPTOPROPENYL/BI
E12	1	MERCAPTOPROPENYLOXY/BI

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E3	0	--> MERCAPTOPROPANYL/CN
E4	3	MERCAPTOPROPIONIC ACID/CN
E5	1	MERCAPTOPROPIONIC ACID, BUTYL ESTER/CN
E6	1	MERCAPTOPROPIONIC ACID, LAURYL ESTER/CN
E7	1	MERCAPTOPROPIONIC ACID, OCTYL ESTER/CN
E8	1	MERCAPTOPROPIONIC ACID-M 90G-PERFLUOROOC
		E TELOMER (GRAFT), ESTER WITH GLYCIDYL METHACRYLAT
E9	1	MERCAPTOPROPIONIC ACID-METHYL METHACRYLATE-M 90G-PERFLUOROOC
		TYLETHYL METHACRYLATE TELOMER (GRAFT), ESTER WITH GLYCIDYL M
		ETHACRYLATE/CN
E10	1	MERCAPTOPROPIONYLAMINOTRIAZOLE/CN
E11	1	MERCAPTOPROPIONYLGLYCINE/CN
E12	1	MERCAPTOPROPYL GROUP-TERMINATED DI-ME SILOXANES/CN

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 CI COM

SH

Me-CH-CO₂H

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L1 3 ANSWERS REGISTRY COPYRIGHT 2003 ACS
 IN Propanoic acid, 3-mercapto- (9CI)
 MF C3 H6 O2 S
 CI COM

HS-CH₂-CH₂-CO₂H

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

L1 3 ANSWERS REGISTRY COPYRIGHT 2003 ACS
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 MF C3 H6 O2 S
 CI IDS, COM

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HO- C CH₂ CH₃

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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2	Apr 08	"Ask CAS" for self-help around the clock
NEWS	3	Jun 03	New e-mail delivery for search results now available
NEWS	4	Aug 08	PHARMAMarketLetter(PHARMAML) - new on STN
NEWS	5	Aug 19	Aquatic Toxicity Information Retrieval (AQUIRE) now available on STN
NEWS	6	Aug 26	Sequence searching in REGISTRY enhanced
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NEWS	9	Sep 16	CA Section Thesaurus available in CAPLUS and CA
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NEWS EXPRESS			April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP),

AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003

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	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

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<http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf>

=> e desmopressin/cn

E1	1	DESMOPLAKIN I (HUMAN KERATINOCYTE GENE DPI REDUCED)/CN
E2	1	DESMOPLAKIN I (HUMAN REDUCED)/CN
E3	1 -->	DESMOPRESSIN/CN
E4	1	DESMOPRESSIN ACETATE/CN
E5	1	DESMOPYRIDINE/CN
E6	1	DESMORAPID/CN
E7	1	DESMORAPID 10/9/CN
E8	1	DESMORAPID 1792/CN
E9	1	DESMORAPID DB/CN
E10	1	DESMORAPID LA/CN
E11	1	DESMORAPID PP/CN
E12	1	DESMORAPID PV/CN

=> s e3

L1 1 DESMOPRESSIN/CN

=> d scan

L1 1 ANSWERS REGISTRY COPYRIGHT 2003 ACS

IN Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine- (9CI)

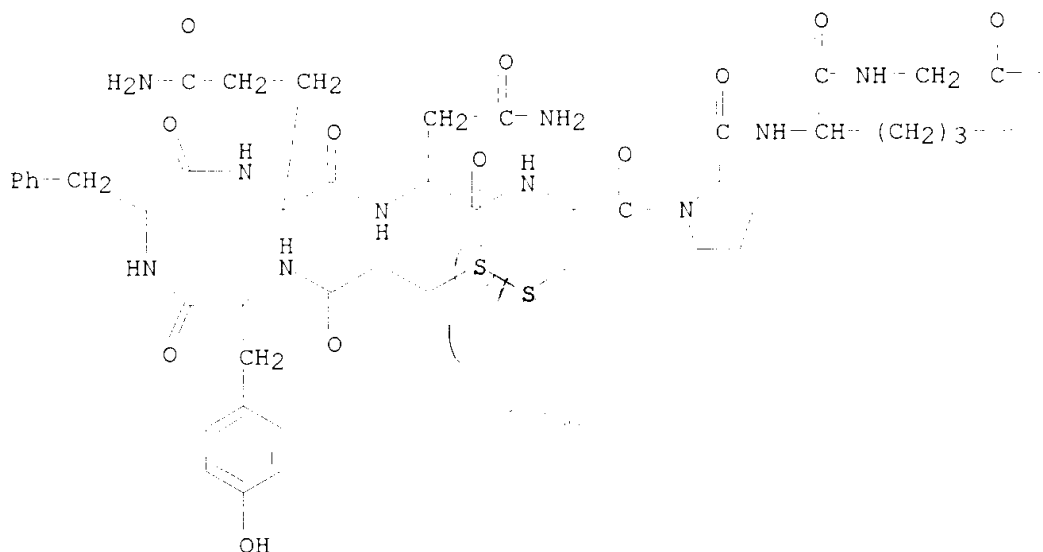
SQL 9

MF C46 H64 N14 O12 S2

CI COM

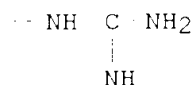
RELATED SEQUENCES AVAILABLE WITH SEQLINK

PAGE 1-A



PAGE 1-B

— NH₂



ALL ANSWERS HAVE BEEN SCANNED

=> s e4

L2 1 "DESMOPRESSIN ACETATE"/CN

=> d scan

L2 1 ANSWERS REGISTRY COPYRIGHT 2003 ACS

IN Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine-, monoacetate (salt) (9CI)

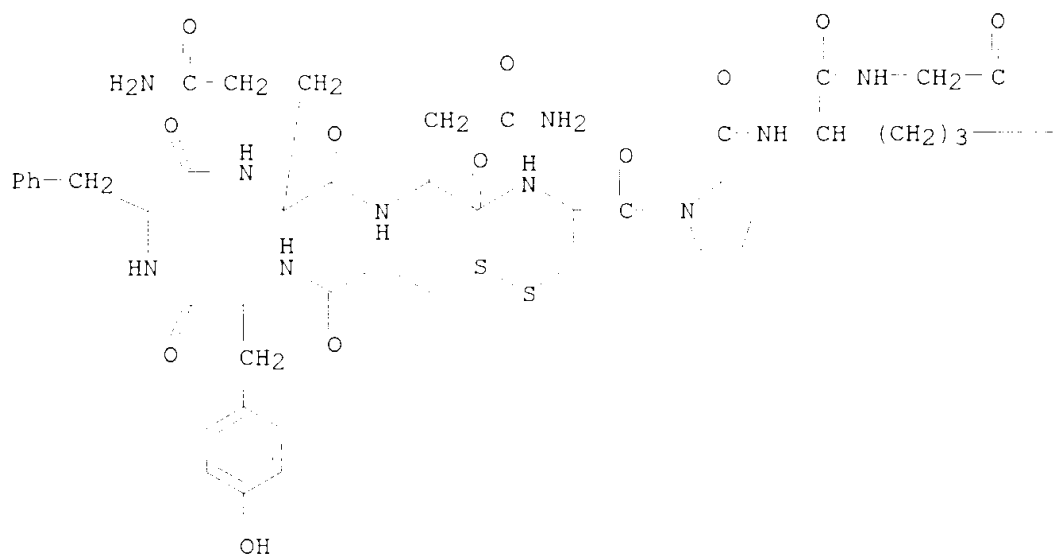
SQL 9
MF C46 H64 N14 O12 S2 . C2 H4 O2

RELATED SEQUENCES AVAILABLE WITH SEQLINK

CM 1

RELATED SEQUENCES AVAILABLE WITH SEQLINK

PAGE 1-A

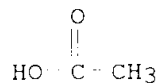


PAGE 1-B

— NH₂

— NH — C — NH₂
|
NH

CM 2



ALL ANSWERS HAVE BEEN SCANNED

=> d

L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS

RN 62288-83-9 REGISTRY
 CN Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine-, monoacetate
 (salt) (9CI) (CA INDEX NAME)
 OTHER CA INDEX NAMES:
 CN 1,2-Dithia-5,8,11,14,17-pentaazacycloeicosane, cyclic peptide deriv.
 OTHER NAMES:
 CN 1-(3-Mercaptopropionic acid)-8-D-arginine vasopressin monoacetate
 CN **Desmopressin acetate**
 CN Octostim
 CN Stimate
 FS PROTEIN SEQUENCE; STEREOSEARCH
 MF C46 H64 N14 O12 S2 . C2 H4 O2
 LC STN Files: ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA,
 CAPLUS, CBNB, CHEMCATS, CIN, DIOGENES, DRUGUPDATES, EMBASE, MRCK*,
 PHARMASEARCH, PROMT, TOXCENTER, USAN, USPAT2, USPATFULL
 (*File contains numerically searchable property data)

RELATED SEQUENCES AVAILABLE WITH SEQLINK

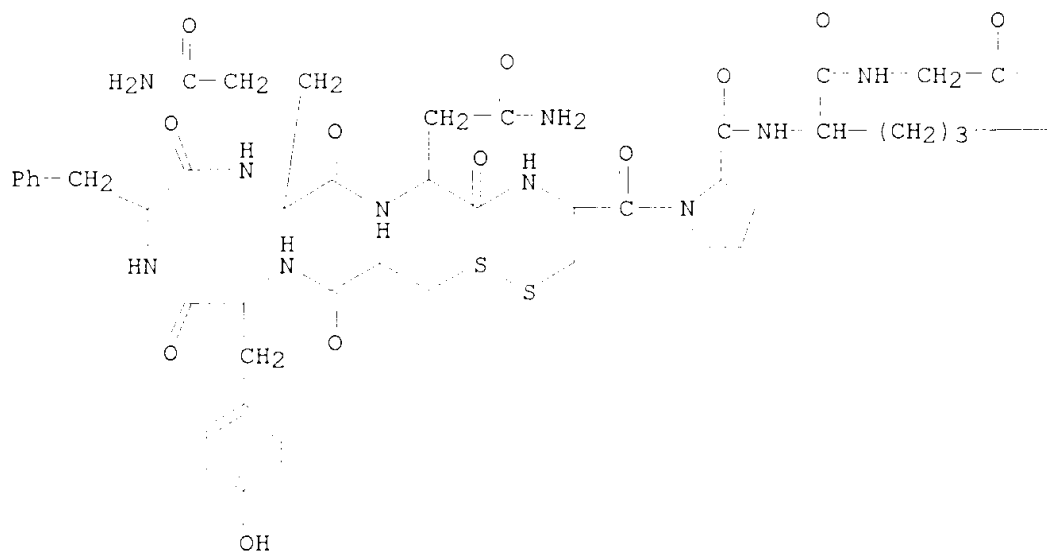
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CRN 16679-58-6

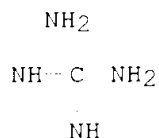
CMF C46 H64 N14 O12 S2

RELATED SEQUENCES AVAILABLE WITH SEQLINK

PAGE 1-A

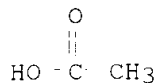


PAGE 1-B



CM 2

CRN 64-19-7
CMF C2 H4 O2



60 REFERENCES IN FILE CA (1957 TO DATE)
60 REFERENCES IN FILE CAPLUS (1957 TO DATE)

=> s e3

L3 1 DESMOPRESSIN/CN

=> d

L3 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS

RN 16679-58-6 REGISTRY

CN Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine- (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 1,2-Dithia-5,8,11,14,17-pentaazacycloeicosane, cyclic peptide deriv.

CN Vasopressin, 1-(3-mercaptopropionic acid)-8-D-arginine- (8CI)

OTHER NAMES:

CN (1-Deamino-8-D-Arg)-vasopressin

CN 1-(3-Mercaptopropionic acid)-8-D-arginine-vasopressin

CN 1-Deamino-1-[D-Arg8]-vasopressin

CN 1-Deamino-[8-D-arginine]vasopressin

CN 1-Desamino-8-D-arginine-vasopressin

CN 1-Desaminocystine-8-D-arginine-vasopressin

CN 8-D-Arginine deaminovasopressin

CN Adiuretin

CN Adiuretin SD

CN DAV Ritter

CN DDAVP

CN **Desmopressin**

CN Desmospray

CN Minirin

CN Minrin

CN [1-β-Mercaptopropionic acid-8-D-arginine]vasopressin

CN [Deaminol-D-arginine8]vasopressin

CN [Desamino-Cys1,D-Arg8]vasopressin

FS PROTEIN SEQUENCE; STEREOSEARCH

DR 57393-40-5, 55479-19-1, 67259-07-8, 74341-59-6, 70368-29-5, 79050-01-4,
81873-22-5, 90242-66-3, 92008-55-4

MF C46 H64 N14 O12 S2

CI COM

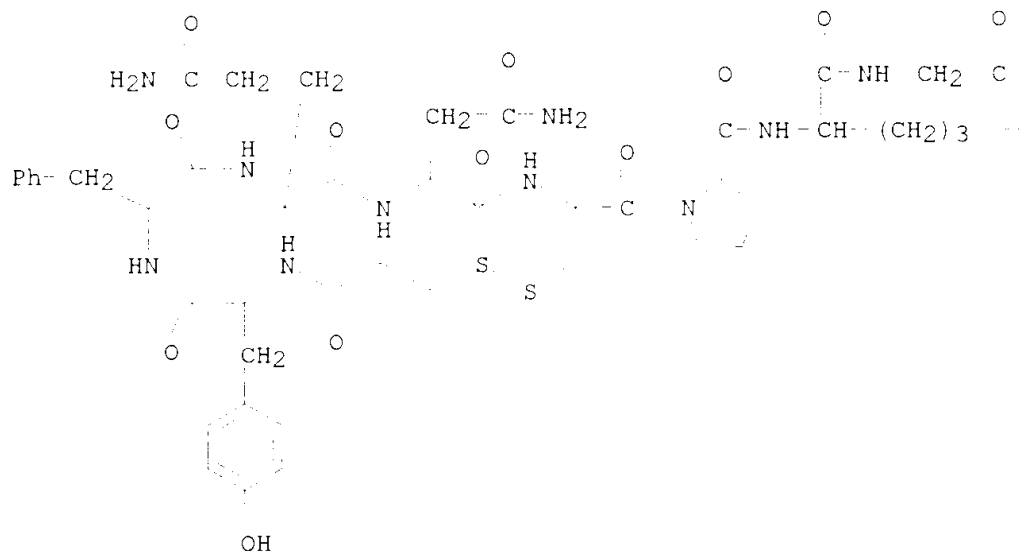
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
BIOTECHNO, CA, CANCERLIT, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST,
CIN, CSCHEM, DDFU, DIOGENES, DRUGNL, DRUGU, DRUGUPDATES, EMBASE, IPA,
MEDLINE, MRCK*, NIOSHTIC, PHAR, PHARMASEARCH, PIRA, PROMT, TOXCENTER,
USAN, USPAT2, USPATFULL, VETU

(*File contains numerically searchable property data)

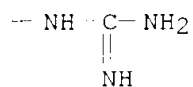
Other Sources: EINECS**, WHO

(**Enter CHEMLIST File for up-to-date regulatory information)

RELATED SEQUENCES AVAILABLE WITH SEQLINK



— NH₂



1143 REFERENCES IN FILE CA (1957 TO DATE)

15 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA

1152 REFERENCES IN FILE CAPLUS (1957 TO DATE)

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LOGINID:SSSPTA1653MXA

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	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

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<http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf>

=> e oxytocin/cn

E1	1	OXYTHIOQUINOX/CN
E2	1	OXYTOCIC/CN
E3	1 -->	OXYTOCIN/CN
E4	1	OXYTOCIN (REDUCED)/CN
E5	1	OXYTOCIN ACETATE (SALT)/CN
E6	1	OXYTOCIN ANTIPARALLEL DIMER/CN
E7	1	OXYTOCIN C-TERMINAL TRIPEPTIDE/CN
E8	1	OXYTOCIN DIACETATE/CN
E9	1	OXYTOCIN DIMERCURY/CN
E10	1	OXYTOCIN FREE ACID/CN
E11	1	OXYTOCIN MONOMERCURY/CN
E12	1	OXYTOCIN PARALLEL DIMER/CN

=> e oxitocin/cn

E1	1	OXITEX 70/CN
E2	1	OXITHIOPURINOL/CN
E3	0 -->	OXITOCIN/CN
E4	1	OXITOL/CN
E5	1	OXITOL ACETATE/CN
E6	1	OXITON TS/CN
E7	1	OXITRIL 4/CN
E8	1	OXITRIPTAN/CN
E9	1	OXITRIPTYLINE/CN
E10	1	OXITROPIUM/CN
E11	1	OXITROPIUM BROMIDE/CN
E12	1	OXIURAN/CN

=> e oxytocin/cn

E1	1	OXYTHIOQUINOX/CN
E2	1	OXYTOCIC/CN
E3	1 -->	OXYTOCIN/CN
E4	1	OXYTOCIN (REDUCED)/CN
E5	1	OXYTOCIN ACETATE (SALT)/CN
E6	1	OXYTOCIN ANTIPARALLEL DIMER/CN
E7	1	OXYTOCIN C-TERMINAL TRIPEPTIDE/CN
E8	1	OXYTOCIN DIACETATE/CN
E9	1	OXYTOCIN DIMERCURY/CN
E10	1	OXYTOCIN FREE ACID/CN
E11	1	OXYTOCIN MONOMERCURY/CN
E12	1	OXYTOCIN PARALLEL DIMER/CN

=> s e3

L1 1 OXYTOCIN/CN

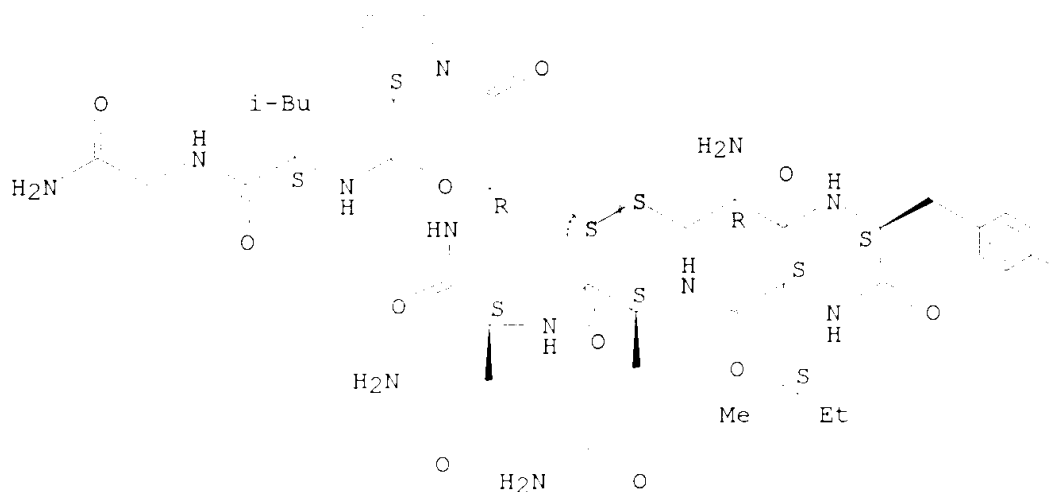
=> d scan

L1 1 ANSWERS REGISTRY COPYRIGHT 2003 ACS
IN **Oxytocin (8CI, 9CI)**
SQL 9
MF C43 H66 N12 O12 S2
CI COM

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Absolute stereochemistry.

PAGE 1-A



PAGE 1-B

OH

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FILE 'HOME' ENTERED AT 09:20:15 ON 16 MAY 2003

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	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'REGISTRY' ENTERED AT 09:20:32 ON 16 MAY 2003
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 DICTIONARY FILE UPDATES: 15 MAY 2003 HIGHEST RN 516445-69-5

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<http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf>

=> fil medline biosis caplus		
COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	1.20	1.41

FILE 'MEDLINE' ENTERED AT 09:22:10 ON 16 MAY 2003

FILE 'BIOSIS' ENTERED AT 09:22:10 ON 16 MAY 2003
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FILE 'CAPLUS' ENTERED AT 09:22:10 ON 16 MAY 2003
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=> e 1-(3-mercaptopropanoic acid)-8-D-argininevasopressin)

E1	1	0ZZX0/BI
E2	13428469	1/BI
E3	0	--> 1-(3-MERCAPTOPROPANOIC ACID)-8-D-ARGININEVASOPRESSIN)/BI
E4	5610906	10/BI
E5	1	10-03-1/BI
E6	3	10-05-4/BI
E7	18	10-10-0/BI
E8	207	10-10-1/BI
E9	150	10-10-2/BI
E10	216	10-10-3/BI
E11	154	10-10-4/BI
E12	270	10-10-5/BI

=> s 1-(3-mercaptopropanoic acid)-8-D-argininevasopressin)

MISSING OPERATOR '1-(3-MERCAPTO'

The search profile that was entered contains terms or nested terms that are not separated by a logical operator.

=> e 1-(3-mercaptopropanoic acid)-8-D-argininevasopressin)/cn

'CN' IS NOT A VALID EXPAND FIELD CODE FOR FILE 'CAPLUS'

E#	FREQUENCY	AT	TERM
--	-----	--	----
E1	0	2	1-(3-MERCAPTO-3-METHYLBUTYRYL)-2-ME-TYR-ARGIPRESSIN/CN
E2	0	2	1-(3-MERCAPTOPROPANOIC ACID)-8-ARG-VASOTOCIN, ACETATE/CN
E3	0	-->	1-(3-MERCAPTOPROPANOIC ACID)-8-D-ARGININEVASOPRESSIN)/CN
E4	0	2	1-(3-MERCAPTOPROPIONIC ACID)-2-(O-ET-TYR)-OXYTOCIN/CN
E5	1	7	1-(3-METHOXY-4-METHYLPHENYL)-2-AMINOPROPANE/CN
E6	0	2	1-(3-METHOXY-4-METHYLPHENYL)-2-AMINOPROPANE, (R)-ISOMER/CN
E7	0	2	1-(3-METHOXY-4-METHYLPHENYL)-2-AMINOPROPANE, (S)-ISOMER/CN
E8	0	2	1-(3-METHOXY-4-TERT-BUTYLDIMETHYLSILYLOXY-5-(2-TERT-BUTYLDIMETHYLSILYLOXY-3-METHOXY-5-HYDROXYMETHYL)PHENYL)PHENYL)-2-(4-FORMYL-2-METHOXYPHENOXY)-1,3-PROPANEDIOL/CN
E9	1		1-(3-METHOXY-4-TERT-BUTYLDIMETHYLSILYLOXY-5-(2-TERT-BUTYLDIMETHYLSILYLOXY-3-METHOXY-5-HYDROXYMETHYL)PHENYL)PHENYL)-2-(4-FORMYL-2-METHOXYPHENOXY)-1,3-PROPANEDIOL/CN
E10	2	7	1-(3-METHYL-2-BUTENYL)-4-(2-(3-HYDROXYPHENYL)-1-PHENYLETHYL)PIPERAZINE/CN
E11	0	2	1-(3-METHYL-2-BUTENYL)-4-(2-(3-HYDROXYPHENYL)-1-PHENYLETHYL)PIPERAZINE DIHYDROCHLORIDE/CN
E12	0	2	1-(3-METHYL-3-PHENYLBUTYL)-4-(2-(3-PYRIDYL)THIAZOLIDIN-4-YLCARBONYL)PIPERAZINE FUMARATE/CN

The indicated field code is not available for EXPAND in this file. To see a list of valid EXPAND field codes, enter HELP SFIELDS at an arrow prompt (=>).

=> s mercaptopropanoic and acid and argininevasopressin

L1 2 MERCAPTOPROPANOIC AND ACID AND ARGinineVASOPRESSIN

=> d

L1 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1978:124051 BIOSIS
DN BA65:11051
TI 1-L-2 HYDROXY-3-MERCAPTO PROPANOIC-**ACID** ANALOGS OF ARGinine
VASOPRESSIN 8-D ARGinine VASOPRESSIN AND 4 VALINE 8-D ARGinine
VASOPRESSIN.
AU LOWBRIDGE J; MANNING M; HALDAR J; SAWYER W

CS DEP. BIOCHEM., MED. COLL. OHIO, TOLEDO, OHIO 43699, USA.
SO J MED CHEM, (1977) 20 (9), 1173-1176.
CODEN: JMCMAR. ISSN: 0022-2623.
FS BA; OLD
LA English

=> d L1

L1 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AN 1978:124051 BIOSIS
DN BA65:11051
TI 1-L-2 HYDROXY-3-MERCAPTO PROPANOIC-**ACID** ANALOGS OF ARGININE
VASOPRESSIN 8-D ARGININE VASOPRESSIN AND 4 VALINE 8-D ARGININE
VASOPRESSIN.
AU LOWBRIDGE J; MANNING M; HALDAR J; SAWYER W
CS DEP. BIOCHEM., MED. COLL. OHIO, TOLEDO, OHIO 43699, USA.
SO J MED CHEM, (1977) 20 (9), 1173-1176.
CODEN: JMCMAR. ISSN: 0022-2623.
FS BA; OLD
LA English

=> d 2 104.39

L1 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
AN 1977:496395 CAPLUS
DN 87:96395
TI [1-(L-2-Hydroxy-3-**mercaptopropanoic acid**)] analogs of
arginine-vasopressin, [8-D-arginine]vasopressin, and [4-valine,8-D-
arginine]vasopressin
AU Lowbridge, John; Manning, Maurice; Haldar, Jaya; Sawyer, Wilbur
CS Dep. Biochem., Med. Coll. Ohio, Toledo, OH, USA
SO Journal of Medicinal Chemistry (1977), 20(9), 1173-6
CODEN: JMCMAR; ISSN: 0022-2623
DT Journal
LA English

=> fil reg

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	17.24	18.65

FILE 'REGISTRY' ENTERED AT 09:29:50 ON 16 MAY 2003
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Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES: 15 MAY 2003 HIGHEST RN 516445-69-5
DICTIONARY FILE UPDATES: 15 MAY 2003 HIGHEST RN 516445-69-5

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2003

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. See HELP PROPERTIES for more information. See STNote 27, Searching Properties in the CAS Registry File, for complete details:
<http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf>

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=> e 1-(3-mercaptopropanoic acid)-8-D-argininevasopressin)/cn
E1      1      1-(3-LAURAMIDOPROPYL)-1,1-DIMETHYLHYDRAZONIUM CHLORIDE/CN
E2      1      1-(3-MERCAPTOPHENYL) ETHANONE/CN
E3      0 --> 1-(3-MERCAPTOPROPANOIC ACID)-8-D-ARGININEVASOPRESSIN)/CN
E4      1      1-(3-MERCAPTOPROPIONIC ACID)-2-L-ALANINEOXYTOCIN/CN
E5      1      1-(3-MERCAPTOPROPIONIC ACID)-2-L-ISOLEUCINEOXYTOCIN/CN
E6      1      1-(3-MERCAPTOPROPIONIC ACID)-6-(3-SELENYL-L-ALANINE) OXYTOCIN
              /CN
E7      1      1-(3-MERCAPTOPROPIONIC ACID)-8-D-ARGININE VASOPRESSIN MONOAC
              ETATE/CN
E8      1      1-(3-MERCAPTOPROPIONIC ACID)-8-D-ARGININE-VASOPRESSIN/CN
E9      1      1-(3-MERCAPTOPROPIONIC ACID) OXYTOCIN/CN
E10     1      1-(3-MERCAPTOPROPYL)-4-METHYLPIPERAZINE/CN
E11     1      1-(3-MERCAPTOPROPYL) SILATRANE/CN
E12     1      1-(3-METHACRYLOXYPROPYL)-3-TRIMETHYLSILYLOXY-1,1,3,3-TETRA-
              METHYLDISILOXANE-METHYL ACRYLATE-METHYL METHACRYLATE-VINYL ACE-
              TATE GRAFT COPOLYMER/CN
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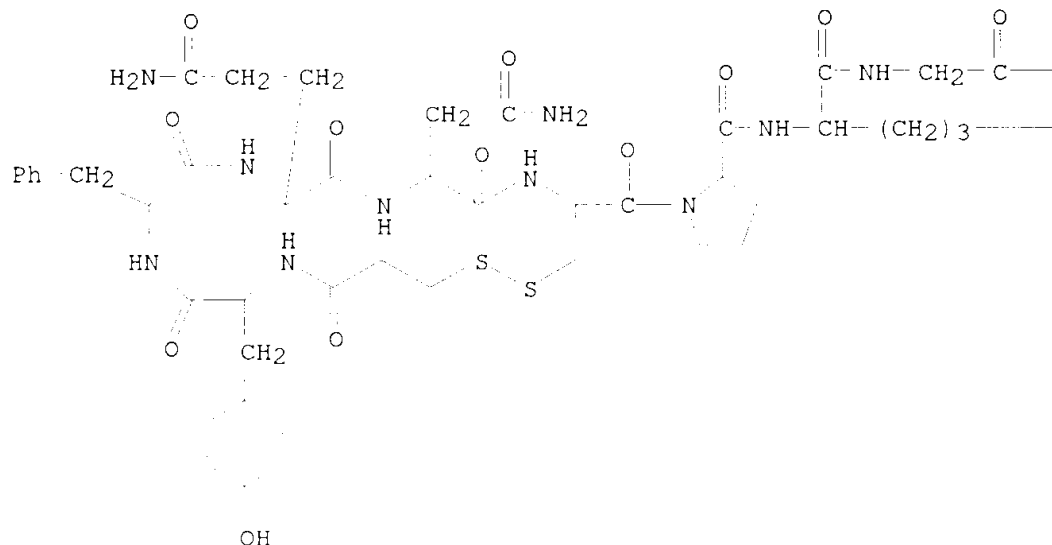
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L2      1 "1-(3-MERCAPTOPROPIONIC ACID)-8-D-ARGININE-VASOPRESSIN"/CN
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=> d scan
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L2      1 ANSWERS   REGISTRY   COPYRIGHT 2003 ACS
IN      Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine- (9CI)
SQL     9
MF      C46 H64 N14 O12 S2
CI      COM
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RELATED SEQUENCES AVAILABLE WITH SEQLINK

PAGE 1-A



-- NH₂

-- NH--C--NH₂
 |
 NH

ALL ANSWERS HAVE BEEN SCANNED

=> s e7

L3 1 "1-(3-MERCAPTOPROPIONIC ACID)-8-D-ARGININE VASOPRESSIN MONOACETATE"/CN

=> d

L3 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS

RN 62288-83-9 REGISTRY

CN Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine-, monoacetate
 (salt) (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 1,2-Dithia-5,8,11,14,17-pentaazacycloeicosane, cyclic peptide deriv.

OTHER NAMES:

CN **1-(3-Mercaptopropionic acid)-8-D-arginine vasopressin monoacetate**

CN Desmopressin acetate

CN Octostim

CN Stimate

FS PROTEIN SEQUENCE; STEREOSEARCH

MF C46 H64 N14 O12 S2 . C2 H4 O2

LC STN Files: ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CBNB, CHEMCATS, CIN, DIOGENES, DRUGUPDATES, EMBASE, MRCK*, PHARMASEARCH, PROMT, TOXCENTER, USAN, USPAT2, USPATFULL
 (*File contains numerically searchable property data)

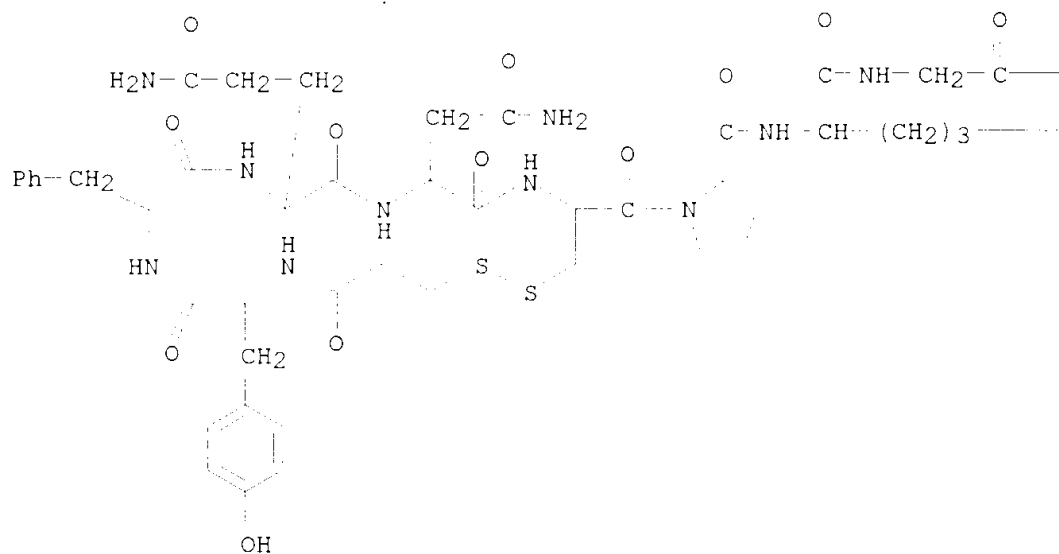
RELATED SEQUENCES AVAILABLE WITH SEQLINK

CM 1

CRN 16679-58-6

CMF C46 H64 N14 O12 S2

RELATED SEQUENCES AVAILABLE WITH SEQLINK



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—NH—C(=NH)—NH₂

CM 2

CRN 64-19-7
CMF C2 H4 O2

HO—C(=O)—CH₃

60 REFERENCES IN FILE CA (1957 TO DATE)
60 REFERENCES IN FILE CAPLUS (1957 TO DATE)

=> s e7

L4 1 "1-(3-MERCAPTOPROPIONIC ACID)-8-D-ARGININE VASOPRESSIN MONOACETATE"/CN

=> d

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2003 ACS
RN 62288-83-9 REGISTRY
CN Vasopressin, 1-(3-mercaptopropanoic acid)-8-D-arginine-, monoacetate (salt) (9CI) (CA INDEX NAME)

OTHER CA INDEX NAMES:

CN 1,2-Dithia-5,8,11,14,17-pentaazacycloeicosane, cyclic peptide deriv.

OTHER NAMES:

CN **1-(3-Mercaptopropionic acid)-8-D-arginine vasopressin monoacetate**

CN Desmopressin acetate

CN Octostim

CN Stimat

FS PROTEIN SEQUENCE; STEREOSEARCH

MF C46 H64 N14 O12 S2 . C2 H4 O2

LC STN Files: ADISNEWS, AGRICOLA, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CAPLUS, CBNB, CHEMCATS, CIN, DIOGENES, DRUGUPDATES, EMBASE, MRCK*, PHARMASEARCH, PROMT, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)

RELATED SEQUENCES AVAILABLE WITH SEQLINK

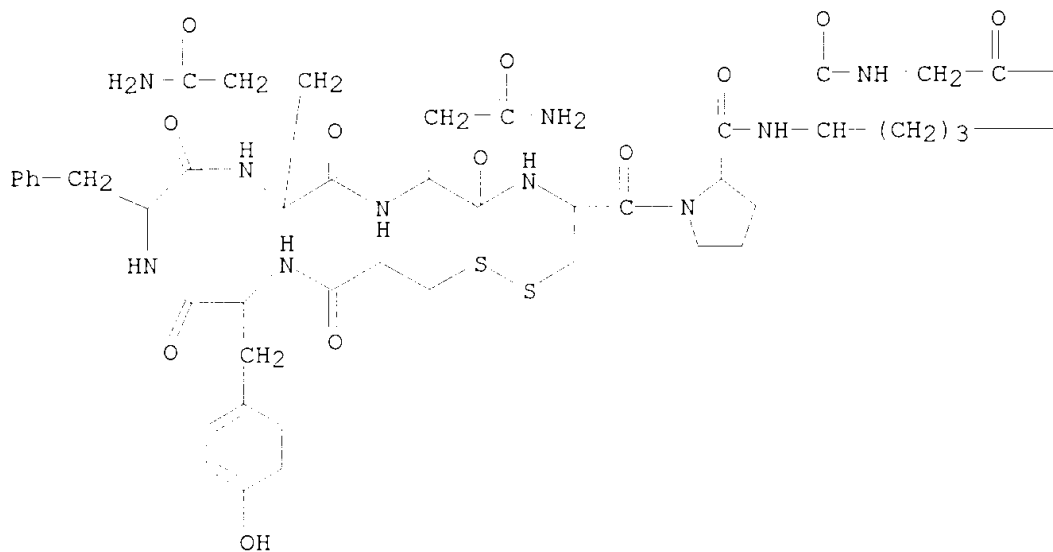
CM 1

CRN 16679-58-6

CMF C46 H64 N14 O12 S2

RELATED SEQUENCES AVAILABLE WITH SEQLINK

PAGE 1-A



PAGE 1-B

NH₂

-- NH C - NH₂

NH

CM 2

CRN 64-19-7
CMF C2 H4 O2

O

HO C-CH₃

60 REFERENCES IN FILE CA (1957 TO DATE)
60 REFERENCES IN FILE CAPLUS (1957 TO DATE)

=> fil medline biosis caplus
COST IN U.S. DOLLARS

SINCE FILE	TOTAL
ENTRY	SESSION
18.02	36.67

FULL ESTIMATED COST

FILE 'MEDLINE' ENTERED AT 09:32:55 ON 16 MAY 2003

FILE 'BIOSIS' ENTERED AT 09:32:55 ON 16 MAY 2003
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FILE 'CAPLUS' ENTERED AT 09:32:55 ON 16 MAY 2003
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COPYRIGHT (C) 2003 AMERICAN CHEMICAL SOCIETY (ACS)

=> s e8

'CN' IS NOT A VALID FIELD CODE

L5 0 "1-(3-MERCAPTOPROPIONIC ACID)-8-D-ARGININE-VASOPRESSIN"/CN

=> s 12

L6 5187 L2

=> dup rem

ENTER L# LIST OR (END):16

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PROCESSING IS APPROXIMATELY 74% COMPLETE FOR L6

PROCESSING IS APPROXIMATELY 99% COMPLETE FOR L6

PROCESSING COMPLETED FOR L6

L7 3718 DUP REM L6 (1469 DUPLICATES REMOVED)

=>

=> s 17 and water and composition and buffer and (sodium chloride)

L8 2 L7 AND WATER AND COMPOSITION AND BUFFER AND (SODIUM CHLORIDE)

=> d

L8 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS

AN 2001:617848 CAPLUS

DN 135:185480

TI Stable nasal, oral and sublingual pharmaceutical preparations containing
desmopressin and malic acid

IN Scheidl, Helmut; Hantich, Gerhard; Hesse, Ernst; Zapf, Thomas

PA Gebro Pharma G.m.b.H., Austria

SO PCT Int. Appl., 26 pp.

CODEN: PIXXD2

DT Patent

LA German

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001060394	A1	20010823	WO 2001-AT7	20010110
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AT 409081	B	20020527	AT 2000-233	20000216
	EP 1255557	A1	20021113	EP 2001-901008	20010110
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	NO 2002003875	A	20021011	NO 2002-3875	20020815
PRAI	AT 2000-233	A	20000216		
	WO 2001-AT7	W	20010110		

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d 2

L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS
AN 2001:581444 CAPLUS
DN 135:157680
TI Pharmaceutical **composition** containing a small or medium size peptide
IN Woodrow, Wayne
PA Patents Exploitation Company B.V., Neth.
SO Eur. Pat. Appl., 12 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1121935	A1	20010808	EP 2001-102385	20010202
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2001233787	A2	20010828	JP 2001-25127	20010201
	US 2001027177	A1	20011004	US 2001-776266	20010202
PRAI	EP 2000-102429	A	20000204		

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d 1-2

L8 ANSWER 1 OF 2 CAPLUS COPYRIGHT 2003 ACS
AN 2001:617848 CAPLUS
DN 135:185480
TI Stable nasal, oral and sublingual pharmaceutical preparations containing desmopressin and malic acid
IN Scheidl, Helmut; Hantich, Gerhard; Hesse, Ernst; Zapf, Thomas
PA Gebro Pharma G.m.b.H., Austria
SO PCT Int. Appl., 26 pp.
CODEN: PIXXD2
DT Patent

LA German

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001060394	A1	20010823	WO 2001-AT7	20010110
	W:	AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM			
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	AT 409081	B	20020527	AT 2000-233	20000216
	EP 1255557	A1	20021113	EP 2001-901008	20010110
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR			
	NO 2002003875	A	20021011	NO 2002-3875	20020815
PRAI	AT 2000-233	A	20000216		
	WO 2001-AT7	W	20010110		
RE.CNT	6	THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT			

L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2003 ACS

AN 2001:581444 CAPLUS

DN 135:157680

TI Pharmaceutical **composition** containing a small or medium size peptide

IN Woodrow, Wayne

PA Patents Exploitation Company B.V., Neth.

SO Eur. Pat. Appl., 12 pp.

CODEN: EPXXDW

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1121935	A1	20010808	EP 2001-102385	20010202
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO			
	JP 2001233787	A2	20010828	JP 2001-25127	20010201
	US 2001027177	A1	20011004	US 2001-776266	20010202
PRAI	EP 2000-102429	A	20000204		
RE.CNT	6	THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT			

=> s 17 and free and preservatives

L9 4 L7 AND FREE AND PRESERVATIVES

=> s 17 and without and preservatives

L10 0 L7 AND WITHOUT AND PRESERVATIVES

=> d 19

L9 ANSWER 1 OF 4 MEDLINE

AN 91262962 MEDLINE

DN 91262962 PubMed ID: 2096493

TI Chlorobutanol, a preservative of desmopressin, inhibits human platelet aggregation and release in vitro.

AU Chen S L; Yang W C; Huang T P; Wann S A; Teng C M

CS Department of Internal Medicine, Veterans General Hospital Taipei, Taiwan,
R.O.C.
SO THROMBOSIS AND HAEMOSTASIS, (1990 Nov 30) 64 (3) 473-7.
Journal code: 7608063. ISSN: 0340-6245.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199107
ED Entered STN: 19910802
Last Updated on STN: 20000303
Entered Medline: 19910715

=> d 19 1-4

L9 ANSWER 1 OF 4 MEDLINE
AN 91262962 MEDLINE
DN 91262962 PubMed ID: 2096493
TI Chlorobutanol, a preservative of desmopressin, inhibits human platelet
aggregation and release in vitro.
AU Chen S L; Yang W C; Huang T P; Wann S A; Teng C M
CS Department of Internal Medicine, Veterans General Hospital Taipei, Taiwan,
R.O.C.
SO THROMBOSIS AND HAEMOSTASIS, (1990 Nov 30) 64 (3) 473-7.
Journal code: 7608063. ISSN: 0340-6245.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199107
ED Entered STN: 19910802
Last Updated on STN: 20000303
Entered Medline: 19910715

L9 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2003 ACS
AN 2001:581444 CAPLUS
DN 135:157680
TI Pharmaceutical composition containing a small or medium size peptide
IN Woodrow, Wayne
PA Patents Exploitation Company B.V., Neth.
SO Eur. Pat. Appl., 12 pp.
CODEN: EPXXDW
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	EP 1121935	A1	20010808	EP 2001-102385	20010202
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2001233787	A2	20010828	JP 2001-25127	20010201
	US 2001027177	A1	20011004	US 2001-776266	20010202
PRAI	EP 2000-102429	A	20000204		
RE.CNT	6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT				

L9 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2003 ACS
AN 2001:300514 CAPLUS
DN 134:331617
TI Oil-in-water emulsion compositions for polyfunctional active ingredients
IN Chen, Feng-jing; Patel, Mahesh V.

PA Lipocine, Inc., USA
SO PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001028555	A1	20010426	WO 2000-US28835	20001018
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	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
	US 2002107265	A1	20020808	US 1999-420159	19991018
PRAI	US 1999-420159	A	19991018		
RE.CNT	6	THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT			

L9 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2003 ACS
AN 2001:136991 CAPLUS
DN 134:198075
TI Triglyceride-**free** compositions and methods for enhanced absorption of hydrophilic therapeutic agents
IN Patel, Mahesh V.; Chen, Feng-Jing
PA Lipocine, Inc., USA
SO PCT Int. Appl., 113 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 7

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001012155	A1	20010222	WO 2000-US18807	20000710
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	RW:	GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG			
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EP	1210063	A1	20020605	EP 2000-947184	20000710
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	US 2001024658	A1	20010927	US 2000-751968	20001229
	US 6458383	B2	20021001		
PRAI	US 1999-375636	A	19990817		
	WO 2000-US18807	W	20000710		
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6391877 54
6420567 54
6432994 54
6458805 54
6465495 54
6444419 54
6465629 54
6350431 54
6406900 54
6471997 54
6410714 54
4761469 53
6473753 53
5554378 52
6303620 52
6399103 52
6294534 52
6350764 52
6410548 52
6472399 52
6410041 52
6268159 52
6296847 52
6337332 52
6338631 52

09776266_QUAL

6407120 52
6420118 52

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Most Frequently Occurring Classifications of Patents Returned
From A Search of 09776266 on March 26, 2003

Original Classifications

4 514/15
2 435/6
2 435/69.1
2 435/7.23
2 514/220
2 514/278
2 536/23.5

Cross-Reference Classifications

6 536/23.1
5 435/320.1
5 530/350
4 530/300
3 424/451
3 424/499
3 424/500
3 514/12
3 514/2
3 514/975
3 530/315
3 530/327
3 536/23.5
3 546/200
2 424/426
2 424/435
2 424/455
2 424/456
2 424/463
2 424/464
2 424/489
2 435/252.3
2 435/325
2 435/401
2 435/7.92
2 514/15
2 514/16
2 514/315
2 514/317
2 514/323
2 514/603
2 514/937
2 514/938
2 514/939

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2 514/940
2 514/941
2 514/942
2 514/943
2 530/328
2 546/17
2 546/18
2 546/201
2 546/290
2 548/247
2 564/86

Combined Classifications

7 536/23.1
6 514/15
5 435/320.1
5 530/350
5 536/23.5
4 424/451
4 530/300
3 424/489
3 424/499
3 424/502
3 435/325
3 435/6
3 435/69.1
3 514/12
3 514/2
3 514/278
3 514/323
3 514/975
3 530/315
3 530/327
3 546/200
2 424/426
2 424/434
2 424/435
2 424/450
2 424/455
2 424/456
2 424/463
2 424/464
2 424/501
2 435/252.3
2 435/4
2 435/471
2 435/7.1
2 435/7.23

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2 435/7.92
2 514/16
2 514/220
2 514/314
2 514/315
2 514/317
2 514/318
2 514/349
2 514/603
2 514/937
2 514/938
2 514/939
2 514/940
2 514/941
2 514/942
2 514/943
2 530/324
2 530/328
2 546/17
2 546/18
2 546/201
2 546/290
2 548/245
2 548/247
2 564/86

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Titles of Most Frequently Occurring Classifications of Patents Returned

From A Search of 09776266 on March 26, 2003

7 536/23.1 (1 OR, 6 XR)
 Class 536 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 536/1.11 .Carbohydrates or derivatives
 536/18.7 ..Nitrogen containing
 536/22.1 ...N-glycosides, polymers thereof, metal
 derivatives (e.g., nucleic acids, oligonuc
 leotides, etc.)
 536/23.1DNA or RNA fragments or modified forms
 thereof (e.g., genes, etc.)

6 514/15 (4 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ
 (DOAI)
 514/2 .Peptide containing (e.g., protein, peptones,
 fibrinogen, etc.) DOAI
 514/15 ..9 to 11 peptide repeating units in known
 peptide chain

5 435/320.1 (0 OR, 5 XR)
 Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY
 435/320.1 VECTOR, PER SE (E.G., PLASMID, HYBRID PLASMID,
 COSMID, VIRAL VECTOR, BACTERIOPHAGE VECTOR,
 ETC.)
 BACTERIOPHAGE VECTOR, ETC.)

5 530/350 (0 OR, 5 XR)
 Class 530 : CHEMISTRY: NATURAL RESINS OR DERIVATIVES;
 PEPTIDES OR PROTEINS; LIGNINS OR REACTION
 PRODUCTS
 THEREOF
 530/350 PROTEINS, I.E., MORE THAN 100 AMINO ACID
 RESIDUES

5 536/23.5 (2 OR, 3 XR)
 Class 536 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 536/1.11 .Carbohydrates or derivatives

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536/18.7 ..Nitrogen containing

536/22.1 ...N-glycosides, polymers thereof, metal
 derivatives (e.g., nucleic acids, oligonu
 cletides, etc.)

536/23.1DNA or RNA fragments or modified forms
 thereof (e.g., genes, etc.)

536/23.5Encodes an animal polypeptide

4 424/451 (1 OR, 3 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS

424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM

424/451 .Capsules (e.g., of gelatin, of chocolate,
 etc.)

4 530/300 (0 OR, 4 XR)
 Class 530 : CHEMISTRY: NATURAL RESINS OR DERIVATIVES;
 PEPTIDES OR PROTEINS; LIGNINS OR REACTION
 PRODUCTS

530/300 THEREOF
 PEPTIDES OF 3 TO 100 AMINO ACID RESIDUES

3 424/489 (1 OR, 2 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS

424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM

424/489 .Particulate form (e.g., powders, granules,
 beads, microcapsules, and pellets)

3 424/499 (0 OR, 3 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS

424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM

424/489 .Particulate form (e.g., powders, granules,
 beads, microcapsules, and pellets)

424/499 ..Contains proteins or derivative or
 polysaccharides or derivative

3 424/502 (0 OR, 3 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS

424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM

424/489 .Particulate form (e.g., powders, granules,

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424/302 beads, microcapsules, and pellets)
...Contains waxes, higher fatty acids, higher
fatty alcohols

2 435/325 (1 OR, 2 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/325 ANIMAL CELL, PER SE (E.G., CELL LINES, ETC.);
COMPOSITION THEREOF; PROCESS OF PROPAGATING
, MAINTAINING OR PRESERVING AN ANIMAL CELL OR COMPOSITION TH
EREOF; PROCESS OF ISOLATING OR SEPARATING AN ANIMAL CELL O
R COMPOSITION THEREOF; PROCESS OF PREPARING A COMPOSITION
CONTAINING AN ANIMAL CELL; CULTURE MEDIA THEREFORE

3 435/4 (2 OR, 1 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/4 MEASURING OR TESTING PROCESS INVOLVING ENZYMES
OR MICRO-ORGANISMS; COMPOSITION OR TEST ST
RIP THEREFORE;
EST STRIP PROCESSES OF FORMING SUCH COMPOSITION OR T
435/6 .Involving nucleic acid

3 435/69.1 (2 OR, 1 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/41 MICRO-ORGANISM, TISSUE CELL CULTURE OR ENZYME
USING PROCESS TO SYNTHESIZE A DESIRED CHEM
ICAL COMPOUND OR COMPOSITION
435/69.1 .Recombinant DNA technique included in method
of making a protein or polypeptide

3 514/12 (3 OR, 3 XR)

Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ

(DOAI)

514/2 .Peptide containing (e.g., protein, peptones,
fibrinogen, etc.) DOAI

514/12 ..25 or more peptide repeating units in known
peptide chain structure

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3 514/2 (0 OR, 3 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ
 G (DOAI)
 514/2 .Peptide containing (e.g., protein, peptones,
 fibrinogen, etc.) DOAI

3 514/278 (2 OR, 1 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ
 G (DOAI)
 514/183 .Heterocyclic carbon compounds containing a
 hetero ring having chalcogen (i.e., O,S,S
 e or Te) or
 nitrogen as the only ring hetero atoms DO
 Ai
 514/277 ..Hetero ring is six-membered consisting of on
 e
 nitrogen and five carbon atoms
 514/278 ...Spiro ring system

3 514/323 (1 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ
 G (DOAI)
 514/183 .Heterocyclic carbon compounds containing a
 hetero ring having chalcogen (i.e., O
 ,S,Se or Te) or
 nitrogen as the only ring hetero atom
 s DOAI
 514/277 ..Hetero ring is six-membered consisting of on
 e
 nitrogen and five carbon atoms
 514/315 ...Piperidines
 514/317Additional ring containing
 514/319The additional ring is one of the cyclos
 in a polycyclo ring system
 514/320Hetero ring in the polycyclo ring system
 514/323Ring nitrogen in the polycyclo ring
 system

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3 514/975 (0 OR, 3 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/975 CHARACTERIZED BY THE DESIGNATED SURFACTANT USE

D

3 530/315 (0 OR, 3 XR)
 Class 530 : CHEMISTRY: NATURAL RESINS OR DERIVATIVES;
 PEPTIDES OR PROTEINS; LIGNINS OR REACTION

PRODUCTS

THEREOF
 530/300 PEPTIDES OF 3 TO 100 AMINO ACID RESIDUES
 530/315 .Oxytocin; vasopressin; related peptides

3 530/327 (0 OR, 3 XR)
 Class 530 : CHEMISTRY: NATURAL RESINS OR DERIVATIVES;
 PEPTIDES OR PROTEINS; LIGNINS OR REACTION

PRODUCTS

THEREOF
 530/300 PEPTIDES OF 3 TO 100 AMINO ACID RESIDUES
 530/327 .11 to 14 amino acid residues in defined
 sequence

3 546/200 (0 OR, 3 XR)
 Class 546 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 546/1 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbons
 546/184 ...Piperidines
 546/192Additional ring containing
 546/195The additional ring is one of the cyclos
 in a polycyclo ring system
 546/196Hetero ring in the polycyclo ring system
 546/200Ring nitrogen in the polycyclo ring
 system

2 424/426 (0 OR, 2 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM
 424/422 .Implant or insert
 424/423 ..Surgical implant or material
 424/426 ...Erodable, resorbable, or dissolving

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- 2 424/434 (1 OR, 1 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM
 424/422 .Implant or insert
 424/434 ..Mucosal (e.g., nasal, etc.)
- 2 424/435 (0 OR, 2 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM
 424/422 .Implant or insert
 424/434 ..Mucosal (e.g., nasal, etc.)
 424/435 ...Mouth
- 2 424/450 (1 OR, 1 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM
 424/450 .Liposomes
- 2 424/455 (0 OR, 2 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM
 424/451 .Capsules (e.g., of gelatin, of chocolate,
 etc.)
 424/455 ..Containing emulsions, dispersions, or
 solutions
- 2 424/456 (0 OR, 2 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
 FORM
 424/451 .Capsules (e.g., of gelatin, of chocolate,
 etc.)
 424/456 ..Gelatin
- 2 424/463 (0 OR, 2 XR)
 Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL

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FORM

424/451 .Capsules (e.g., of gelatin, of chocolate,
etc.

424/463 ..Coated capsules

2 424/464 (0 OR, 2 XR)

Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
FORM

424/464 .Tablets, lozenges, or pills

2 424/501 (1 OR, 1 XR)

Class 424 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

424/400 PREPARATIONS CHARACTERIZED BY SPECIAL PHYSICAL
FORM

424/489 .Particulate form (e.g., powders, granules,
beads, microcapsules, and pellets)

424/501 ..Contains solid synthetic resin

2 435/252.3 (0 OR, 2 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/243 MICRO-ORGANISM, PER SE (E.G., PROTOZOA, ETC.);
COMPOSITIONS THEREOF; PROCES OF PROPAGATI

NG, MAINTAINING OR

S THEREOF; PROCESS

ONTAINING A

PRESERVING MICRO-ORGANISMS OR COMPOSITION

OF PREPARING OR ISOLATING A COMPOSITION C

MICRO-ORGANISM; CULTURE MEDIA THEREFOR

435/252.1 .Bacteria or actinomycetales; media therefor

435/252.3 ..Transformants (e.g., recombinant DNA or
vector or foreign or exogenous gene contain

ing, fused

bacteria, etc.)

2 435/4 (1 OR, 1 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/4 MEASURING OR TESTING PROCESS INVOLVING ENZYMES
OR MICRO-ORGANISMS; COMPOSITION OR TEST STR

IP THEREFORE;

ST STRIP

PROCESSES OF FORMING SUCH COMPOSITION OR TE

2 435/471 (0 OR, 2 XR)

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Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/455 .Introduction of a polynucleotide molecule into
an animal cell
435/471 .Introduction of a polynucleotide molecule into
a microorganism
(e.g., bacteria, protozoa, bacteriophage, etc.)

2 435/7.1 (1 OR, 1 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/4 MEASURING OR TESTING PROCESS INVOLVING ENZYMES
OR MICRO-ORGANISMS; COMPOSITION OR TEST STRIP
THEREFORE;
PROCESSES OF FORMING SUCH COMPOSITION OR TEST STRIP
435/7.1 .Involving antigen-antibody binding, specific
binding protein assay or specific ligand-receptor binding
assay

2 435/7.23 (2 OR, 0 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

435/4 MEASURING OR TESTING PROCESS INVOLVING ENZYMES
OR MICRO-ORGANISMS; COMPOSITION OR TEST STRIP
THEREFORE;
PROCESSES OF FORMING SUCH COMPOSITION OR TEST STRIP
435/7.1 .Involving antigen-antibody binding, specific
binding protein assay or specific ligand-receptor binding
assay
435/7.2 ..Involving a micro-organism or cell membrane
bound antigen or cell membrane bound receptor or cell
membrane bound antibody or microbial lysate
435/7.21 ...Animal cell
435/7.23Tumor cell or cancer cell

2 435/7.92 (2 OR, 2 XR)

Class 435 : CHEMISTRY: MOLECULAR BIOLOGY AND MICROBIOLOGY

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435/4 MEASURING OR TESTING PROCESS INVOLVING ENZYMES
OR MICRO-ORGANISMS; COMPOSITION OR TEST
STRIP THEREFORE;
TEST STRIP PROCESSES OF FORMING SUCH COMPOSITION OR
435/7.1 .Involving antigen-antibody binding, specific
binding protein assay or specific ligand-
receptor binding assay
435/7.9 ..Assay in which an enzyme present is a label
435/7.92 ...Heterogeneous or solid phase assay system
(e.g., ELISA, etc.)

2 514/16 (2 OR, 0 XR)
Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS
514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ
G
(DOAI)
514/2 .Peptide containing (e.g., protein, peptones,
fibrinogen, etc.) DOAI
514/16 ..7 or 8 peptide repeating units in known
peptide chain

2 514/220 (2 OR, 0 XR)
Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS
514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ
G
(DOAI)
514/168 .Heterocyclic carbon compounds containing a
hetero ring having chalcogen (i.e., O,S,
Se or Te) or
nitrogen as the only ring hetero atoms
OAI
514/218 ..Hetero ring is seven-membered consisting of
two nitrogens and five carbon atoms
514/219 ...Polycyclo ring system having the
seven-membered hetero ring as one of the c
yclos
514/220Tricyclo ring system having the
seven-membered hetero ring as one of the cy
clos

2 514/314 (1 OR, 1 XR)
Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING

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COMPOSITIONS

514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ

(DOAI)

514/183 .Heterocyclic carbon compounds containing a
hetero ring having chalcogen (i.e., O,
S, Se or Te) or
nitrogen as the only ring hetero atoms

514/277 ..Hetero ring is six-membered consisting of on
nitrogen and five carbon atoms

514/279 ...Polycyclo ring system having the
six-membered hetero ring as one of the c

514/299Bicyclo ring system having the six-membere
hetero ring as one of the cyclos

514/311Quinolines (including hydrogenated)

514/314Additional hetero ring attached directly
or indirectly to the quinoline ring system

by nonionic

bonding

2 514/315 (0 OR, 2 XR)

Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ

(DOAI)

514/183 .Heterocyclic carbon compounds containing a
hetero ring having chalcogen (i.e., O,S,S
e or Te) or
nitrogen as the only ring hetero atoms DO

514/277 ..Hetero ring is six-membered consisting of on
nitrogen and five carbon atoms

514/315 ...Piperidines

2 514/317 (0 OR, 2 XR)

Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ

(DOAI)

514/183 .Heterocyclic carbon compounds containing a
hetero ring having chalcogen (i.e., O,S,

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Se or Te, or

nitrogen as the only ring hetero atoms D

OAI

514/277 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbon atoms

514/315 ...Piperidines

514/317Additional ring containing

2 514/318 (1 OR, 1 XR)

Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ

G

(DOAI)

514/183 .Heterocyclic carbon compounds containing a
hetero ring having chalcogen (i.e., O,S

,Se or Te) or

nitrogen as the only ring hetero atoms

DOAI

514/277 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbon atoms

514/315 ...Piperidines

514/317Additional ring containing

514/318The additional ring is a six-membered
hetero ring consisting of one nitrogen and

five carbon

atoms

2 514/349 (1 OR, 1 XR)

Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
COMPOSITIONS

514/1 DESIGNATED ORGANIC ACTIVE INGREDIENT CONTAININ

G

(DOAI)

514/183 .Heterocyclic carbon compounds containing a
hetero ring having chalcogen (i.e., O,S,

Se or Te) or

nitrogen as the only ring hetero atoms D

OAI

514/277 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbon atoms

514/345 ...Chalcogen bonded directly to ring carbon of
the six-membered hetero ring514/349Nitrogen attached directly to the
six-membered hetero ring by nonionic bondin

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g

- 2 514/603 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/266.24The additional hetero ring consists of
 carbon and chalcogen as the only ring
 members
 514/578 .Nitrogen containing other than solely as a
 nitrogen in an inorganic ion of an addit
 ion salt, a nitro
 or a nitroso DOAI
 514/601 ..Sulfonamides (i.e., Q-(O=)S(=O)-N, wherein Q
 is a substituent and wherein any substitu
 ent attached to
 the nitrogen will be referred to as E)
 514/602 ...Q contains benzene ring
 514/603Nitrogen in Q
- 2 514/937 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/937 DISPERSION OR EMULSION
- 2 514/938 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/937 DISPERSION OR EMULSION
 514/938 .Oil-water type
- 2 514/939 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/937 DISPERSION OR EMULSION
 514/938 .Oil-water type
 514/939 ..Mineral oil-water type
- 2 514/940 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/937 DISPERSION OR EMULSION
 514/938 .Oil-water type
 514/939 ..Mineral oil-water type
 514/940 ...Quick break type
- 2 514/941 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS

514/937 DISPERSION OR EMULSION
 514/938 .Oil-water type
 514/939 ..Mineral oil-water type
 514/941 ...Polyoxyalkylated compound containing

2 514/942 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/937 DISPERSION OR EMULSION
 514/938 .Oil-water type
 514/939 ..Mineral oil-water type
 514/942 ...Organic sulfonate, sulfate or sulfite
 containing

2 514/943 (0 OR, 2 XR)
 Class 514 : DRUG, BIO-AFFECTING AND BODY TREATING
 COMPOSITIONS
 514/937 DISPERSION OR EMULSION
 514/938 .Oil-water type
 514/939 ..Mineral oil-water type
 514/943 ...Higher fatty acid or derivative containing

2 530/324 (1 OR, 1 XR)
 Class 530 : CHEMISTRY: NATURAL RESINS OR DERIVATIVES;
 PEPTIDES OR PROTEINS; LIGNINS OR REACTION

PRODUCTS

THEREOF
 530/300 PEPTIDES OF 3 TO 100 AMINO ACID RESIDUES
 530/324 .25 or more amino acid residues in defined
 sequence

2 530/328 (0 OR, 2 XR)
 Class 530 : CHEMISTRY: NATURAL RESINS OR DERIVATIVES;
 PEPTIDES OR PROTEINS; LIGNINS OR REACTION

PRODUCTS

THEREOF
 530/300 PEPTIDES OF 3 TO 100 AMINO ACID RESIDUES
 530/328 .8 to 10 amino acid residues in defined
 sequence

2 546/17 (0 OR, 2 XR)
 Class 546 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 546/1 ..Hetero ring is six-membered consisting of on
 e nitrogen and five carbons
 546/15 ...Spiro

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546/16The spiro includes the six-membered hetero ring

546/17Polycyclo ring system having one of the two rings which form the spiro as one of th

e cyclos

147718 (0 OR, 1 XR)

Class 546 : ORGANIC COMPOUNDS -- PART OF THE CLASS
532-570 SERIES

546/1 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbons

546/15 ...Spiro

546/16The spiro includes the six-membered hetero ring

546/17Polycyclo ring system having one of the two rings which form the spiro as one of t

he cyclos

546/18Polycyclo ring system having the six-membered hetero ring as one of the cycl

os

2 546/201 (0 OR, 2 XR)

Class 546 : ORGANIC COMPOUNDS -- PART OF THE CLASS
532-570 SERIES

546/1 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbons

546/184 ...Piperidines

546/192Additional ring containing

546/195The additional ring is one of the cyclos in a polycyclo ring system

546/196Hetero ring in the polycyclo ring system

546/200Ring nitrogen in the polycyclo ring system

546/201Bicyclo ring system which is indole (including hydrogenated)

2 546/290 (0 OR, 2 XR)

Class 546 : ORGANIC COMPOUNDS -- PART OF THE CLASS
532-570 SERIES

546/1 ..Hetero ring is six-membered consisting of on

e

nitrogen and five carbons

546/290 ...Chalcogen bonded directly to ring carbon of the six-membered hetero ring

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2 548/245 (1 OR, 1 XR)
 Class 548 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 548/100 ..Hetero ring is five-membered having two or
 more ring hetero atoms of which at least
 one is nitrogen
 (e.g., selenazoles, etc.)
 548/240 ...1,2-oxazoles (including hydrogenated)
 548/245Nitrogen bonded directly to ring carbon of
 the oxazole ring

2 548/247 (0 OR, 2 XR)
 Class 548 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 548/100 ..Hetero ring is five-membered having two or
 more ring hetero atoms of which at least
 one is nitrogen
 (e.g., selenazoles, etc.)
 548/240 ...1,2-oxazoles (including hydrogenated)
 548/247Plural double bonds between ring members o
 f
 the oxazole ring

2 564/86 (0 OR, 2 XR)
 Class 564 : ORGANIC COMPOUNDS -- PART OF THE CLASS
 532-570 SERIES
 564/1 .AMINO NITROGEN CONTAINING (E.G., UREA,
 SULFONAMIDES, NITROSAMINES, OXYAMINES, E
 TC., AND SALTS
 THEREOF)
 564/80 ..Sulfonamides (i.e., Q-(O=)S(=O)-HNH, wherein
 Q is a substituent and wherein any substi
 tuent replacing
 one or both hydrogens shown will be refer
 red to as E)
 564/84 ...Substituent Q contains benzene ring
 564/86Nitrogen in substituent Q

ab 2
 able 1
 about 1
 absolute 1
 abstract 1
 adaptable 2
 accomplishment 1
 according 1
 accordingly 1
 acetate 1
 acid 7
 acting 1
 active 6
 activity 5
 actuator 1
 addition 1
 additive 1
 additives 2
 administered 1
 administering 1
 administration 2
 adsorbed 2
 adsorption 16
 advantages 1
 after 1
 agent 3
 aim 1
 air 1
 all 1
 allergic 1
 allowing 1
 already 1
 also 2
 always 1
 amine 3
 among 2
 amount 3
 amounts 1
 an 12
 ana 1
 anaesthesiology 1
 analogous 1
 analogues 4
 and 36
 antidiuretic 1
 antimicrobial 1
 antioxidants 1
 anyway 1

apparent 2
application 5
applications 1
aqueous 2
ar 1
are 12
arginin 2
argininevasopressin 2
arising 1
art 3
arts 1
as 22
asma 1
aspiration 1
ass 1
associated 1
astic 2
at 7
atoms 4
attesting 1
attwood 1
auto 1
avoiding 1
bacteria 1
banning 1
be 4
because 2
been 3
before 3
being 1
benza 1
benzalkonium 2
besides 1
between 1
biologic 1
blocking 1
bonds 1
both 1
bottles 1
bridges 1
buffer 5
but 1
by 12
ca 1
calcitonin 1
capable 1
case 1
cause 1

characterized 1
chloride 5
chlorobutanol 1
ciliar 1
cilia 1
citrate 2
citric 5
claim 1
claims 2
clinical 1
cm 1
complete 1
composition 21
compositions 7
comprised 1
concerned 2
considerable 1
consisting 1
contain 2
container 6
containing 14
contains 4
contamination 1
context 1
control 1
controlling 2
cross 1
cunningham 1
cyc 1
cyclic 5
cyclus 3
deamino 2
degradation 5
demonstrated 1
derivatives 5
desamine 1
described 1
description 2
depression 3
device 4
diabetes 1
dihydrate 4
discloses 1
disinfectant 2
disodium 3
disorders 1
do 1
does 2

drawback 2
drug 4
drugs 3
due 1
during 3
easily 1
easy 1
edition 1
effective 1
embodiment 2
embraces 2
encountered 1
endowed 1
entailing 1
enuresis 1
environment 2
envisaged 1
ep 2
epitomising 1
equipped 1
erich 1
especially 2
esters 1
et 1
even 2
examp 2
example 7
examples 2
excipients 1
exhibiting 1
extended 1
fact 1
far 2
february 1
ferring 2
field 1
filed 1
filled 1
filter 1
filtered 1
filters 1
fl 1
florence 1
for 10
foregoing 1
formulations 1
found 1
free 7

from 22
further 2
furthermore 1
general 1
giving 2
glass 2
gmbh 1
goals 1
group 1
half 1
has 4
have 2
hereby 1
hfe 1
hofmann 1
nours 1
human 2
hydrate 1
ihydrate 1
im 1
in 23
incorporated 1
ing 1
inhibiting 1
inhibitor 1
inhibitors 2
insipidus 1
insulin 2
intended 1
intravenous 1
invention 17
investigated 1
investigations 1
io 3
irreversible 1
irritative 1
is 22
it 3
its 1
jo 1
kept 1
kind 3
known 2
konium 1
laboratory 1
least 4
life 1
like 2

likewise 1
long 1
lose 1
loss 2
losses 2
lu 1
maintained 1
maintaining 1
many 2
marketable 1
martindale 1
material 3
materials 1
may 3
mechanism 1
medium 14
mercaptopropanoic 2
mercaptopropanyl 1
methyl 1
mg 10
ml 2
monocarba 1
monohydrate 1
more 6
most 5
motility 1
mucosae 1
multidose 1
name 3
nasal 3
nitrogen 1
nocturnal 1
not 4
now 2
number 1
nvent 1
occur 1
of 98
often 3
oftheart 1
egues 1
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ne 3
ongoing 1
only 1
onto 3
or 29
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osmotic 1
over 1
overcoming 1
ow 1
oxftocin 1
oxidizab 1
oxitocin 2
particu 1
particular 4
particularly 1
patent 3
peptide 14
peptides 11
petty 1
pfeiffer 1
ph 2
pharmaceutica 1
pharmaceutical 11
pharmaceutically 3
pharmacy 1
phosphate 3
physicochemical 1
physiologic 1
physiological 1
plasma 1
plastic 1
point 1
polymeric 2
polypropylene 1
polystyrene 1
positive 1
possibility 1
possible 1
potency 4
powerful 2
pp 2
pre 1
preferab 2
preferably 11
preferred 7
prejudice 1
preparation 1
prepared 2
presence 1
present 14
preservative 6
preservatives 10
preserve 1

press 1
pressure 1
prevent 1
preventing 2
prevention 2
principle 6
principles 1
prior 3
priority 1
problem 3
problems 1
process 1
products 1
protection 1
protein 1
provided 2
pump 1
purified 1
quaternary 3
quickly 1
radical 1
rhetocin 1
reactions 1
ready 1
rebound 1
recently 2
reference 3
related 1
relates 2
remarkable 1
report 1
reported 2
represents 1
required 2
reviewed 1
room 2
salts 2
scale 1
seeming 1
selected 2
sets 2
shall 1
shelf 2
short 1
show 2
shows 1
size 14
small 1

small 14
sodium 2
solution. 4
solutions 5
source 1
specific 1
spray 3
springer 1
stab 1
stable 2
state 1
sterile 2
sterilely 1
substances 1
such 16
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suggested 1
suitable 1
suitably 1
sulfur 6
summary 1
suppression 1
surface 1
surprising 1
temperature 2
terlipressin 1
term 3
termed 1
test 1
that 1
thanks 1
that 11
the 126
their 1
therapeutic 1
therapeutically 1
therapy 1
therefore 1
thereof 4
these 3
they 2
third 1
this 5
those 5
though 2
through 1
this 1
time 1

titre 2
to 17
toxicological 1
treatment 1
trials 1
triglycin 1
trisod 1
trisodium 1
tubes 1
tubing 1
two 2
tyrosine 1
under 1
unlike 1
urinary 1
us 1
use 5
used 1
useful 1
utterly 1
value 1
values 2
vasopressin 6
verlad 1
very 1
vial 1
view 1
walls 1
walls 5
water 2
weh 1
well 1
when 1
which 4
will 1
with 4
within 4
wo 3
ysin 1